Advancing Clinical practice in the management of Deep Vein Thrombosis (DVT).

Development, application and evaluation of the AUTAR DVT scale.

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Doctor of Philosophy

2002

Advancing clinical practice in the management of Deep Vein Thrombosis (DVT). Development, application and evaluation of the Autar DVT risk assessment scale

Volume 1 of 2

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August 2002.

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Acknowledgements

Many colleagues have been instrumental in enabling me to complete the study. I am grateful to Professor Mel Chevannes, who in her capacity as my first supervisor and Director of Study, constantly enquired into my progress and provided the necessary support.

Unreserved thanks are due to Dr F Dewhurst, Visiting Research Fellow, who was my second supervisor. I am indebted to him for the regular supervision and feedback in relation to the study.

Dr N Longford, Senior Research Fellow, made significant contribution to my study, as another second supervisor, in guiding me through the statistical components of the study.

Thanks go to my wife Cathy and my three children, Rebecca, Christopher and Richard for their continued tolerance and forbearance.

Finally, I convey my sincere thanks to the clinical colleagues who were generous with their time and so ably collected data prospectively for the study.

Key to Abbreviations

ANS	Anticoagulant Nurse Specialist
APCR	Activated Protein C Resistance
APTT	Activated Partial ThromboplastinTime
BMA	British Medical Association
BMI	Body Mass Index
BNF	British National Formulary
CI	Confidence Interval
CVA	Cerebro Vascular Accident
DoH	Department of Health
DVT	Deep Vein Thrombosis
ELT	Euglobulin Lysis Time
ENB	English National Board
EUA	Examination Under Anaesthesia
FDP	Fibrinogen Degradation Products
FN	False Negative
FP	False Positive
GCS	Graduated Compression Stockings
HITT	Heparin Induced Thrombocytopenia Thrombosis
HLP	Higher Level of Practice
HMSO	Her Majesty's Stationery Office
HRT	Hormone Replacement Therapy
HTD	Hereditary Thrombotic Disease
IBD	Inflammatory Bowel Disease
ICC	Intra-class Correlation Coefficients
ICD	International Classification of Diseases
INR	International Normalised Ratio
IPC	Intermittent Pneumatic Compression
LL	Log likelihood
LMWH	Low Molecular Weight Heparin
MAU	Medical Assessment Unit
NCPOD	National Confidential Enquiry into Peri-Operative Deaths
MI	Myocardial Infarction

NHS	National Health Service
NIH	National Institutes of Health
NMC	Nursing & Midwifery Council
NPV	Negative Positive Value
OHE	Office of Home Economics
OPCS	Office of Population Censuses and Surveys
PAI	Plasminogen Activator Inhibitor
PE	Pulmonary Embolism
PPS	Post Phlebitis Syndrome
PPV	Positive Predictive Value
PREP	Post Registration Education and Practice
PSG	Polycythaemia Study Group
PT	Prothrombin Time
ROC	Receiver Operating Characteristic
SIGN	Scottish Intercollegiate Guidelines Network
Sig	Significance
THRiFT	Thromboembolic Risk Factors
TN	True Negative
TP	True Positive
t-PA	Tissue- Plasminogen Activator
UFH	Unfractionated Heparin
UKCC	United Kingdom Central Council

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Abstract

Deep Vein Thrombosis (DVT) is a disease of hospitalised patients and is a precursor of Pulmonary Embolism (PE), a potentially fatal complication. DVT and PE are preventable and venous thromboprophylaxis consensus groups recommend that patients be risk assessed and accordingly receive appropriate prophylaxis.

The Scope of Professional Practice (UKCC, 1992) enables nurses with appropriate knowledge and clinical competence to explore new territories, previously the exclusive province of doctors. In the spirit of the position statement of professional practice framework, the Autar DVT scale (1994) was developed to identify patients at risk, so that appropriate venous thromboprophylaxis can be initiated. The scale is composed of seven categories of risk factors derived from Virchow's triad in the genesis of DVT.

In this study, the DVT scale was re-validated on 150 patients across three distinct clinical specialities in order to allow for generalisation of the findings. DVT is a continuing problem and for this significant reason, the patients were followed up for a minimum of three months after discharge from hospital. Interestingly, 39 per cent of the patients with DVT (11/28) developed this insidious condition at home. Five reproducibility studies on the orthopaedic, medical and surgical directorates achieved kappa values ranging between 0.88 to 0.95, confirming the consistency of the instrument. A Receiver Operating Characteristic (ROC) curve was constructed to determine the optimal predictive accuracy of the DVT scale and a cutoff score of 11 yielded approximately 70 per cent sensitivity. Data from two patients, who could not be followed up, on account of discharge to no fixed abodes, were excluded for the sensitivity analysis of the DVT scale. Overall, 115 patients out of the 148 (78%) were correctly classified.

However, the administration of venous thromboprophylaxis masked the predictive efficiency of the DVT scale in relation to its sensitivity and specificity. A high predictive value of 84 per cent of negative outcome was achieved at the cost of a predictive value of 37 per cent of positive outcome. The DVT scale exhibited good clinical and practical application. Data extrapolation also suggests that although the clinical areas use some venous thromboprophylaxis strategies, in practice they are not consistently applied.

Chapter One

INTRODUCTION

Scope of Professional Practice

The healthcare needs of the nation are forever changing. As a result, the nature, organisation and delivery of healthcare have evolved in response to the challenge. In order to meet the changing needs of patients, the Scope of Professional Practice (UKCC, 1992) enables nurses with the appropriate knowledge, skills and competence to boldly explore new territories that were previously the exclusive province of doctors. However, registered nurses

"must ensure that any enlargement or adjustment of the scope of personal professional practice must be achieved without compromising or fragmenting existing aspects of professional practice and care"

UKCC, 1992.

Clinical skills such as intravenous drug administration, cannulation, venepuncture and endoscopic examination have become commonplace and subsumed into the core of nursing practice and education.
An innovative and groundbreaking Post Registration Education and Practice (PREP) framework was launched in 1994 and UKCC agreed that:

"Specialist practitioners will demonstrate higher levels of clinical decision making and will be able to monitor and improve standards of care through supervision of practice, clinical nursing audit, developing and leading practice, contributing to research, teaching and supporting professional colleagues"

UKCC, 1994: PREP 9:47, 28:10.

Nurses are no longer the "mechanical hands of doctors" (Castledine, 1996) but are now recognised as an equal partner in the delivery of holistic care. The rapidly evolving environment of heath care provision has witnessed the emergence of nurses working at a Higher Level of Practice (HLP). Developments in the structure, funding and organisation of health care will continue to offer new challenges and opportunities to nurse practitioners. In some NHS trusts, advanced neonatal practitioners are replacing junior doctors in special care baby units, surgical nurse practitioners run pre-

admission clinics and organise theatre lists (Tuthill, 1995; Dowling et al, 1996). In 1997, the government had signalled its wish to:

"extend the recent developments in the roles of nurses working in acute and community services. The government is committed to encouraging and supporting the development of nursing practice in these ways"

(The New NHS. Modern. Dependable,1997).

Similar initiatives have led to the setting up of nurse-led anticoagulant clinics (Brown, 1998) and a nurse-led outpatient anticoagulant clinic (Pout, 1999). Traditionally, such departments have been managed exclusively by consultant haematologist services, with little or no involvement of specialist nurses. Historically, nurse led anticoagulant clinics have emanated from studies in USA managed by an Anticoagulant Nurse Specialist (ANS). Taylor et al (1997) evaluated the cost and effectiveness of ANS services and concluded that they are a viable alternative model of anticoagulant service provision. Such services have a number of advantages over consultant haematologist services. As there is an increase in the number of patients being referred for life long treatment, ANS are able to provide domiciliary services for the housebound. Patient's satisfaction is increased and the services provided by ANS are very acceptable to GPs. They also ensure a reduction in the quantity of drugs that might interact with the homeostatic mechanisms (Van de Pette & Mackie, 1996).

Indeed, it is within the scope of the professional practice development framework that DVT as an area of recognised and growing concern across diverse patient groups was explored as illustrated in table 1.1, highlighting the scale of the problem. A DVT risk assessment tool was developed (Autar, 1994) to facilitate its proactive and comprehensive management by nurses, doctors and other members of the multidisciplinary team, in particular the physiotherapists and occupational therapists.

Table 1.1: Risk level by patient group

Speciality	Incidence of DVT (weighted mean) %
General surgery	25
Orthopaedic surgery	45-51
Urology	9-32
Gynaecological surgery	14-22
Neurosurgery including strokes	22-56
Multiple Trauma	50
General Medical	17

Source: International Consensus Statement, 1997.

Deep Vein Thrombosis: scale of the problem.

Deep vein thrombosis (DVT), as the term implies, is the formation of a clot in the deep vein. The deep veins are so-called because they are embedded intramuscularly in the deep fascia, in contrast to the superficial veins that are extramuscularly, outside the deep fascia (Autar 1996 c). While deep veins are present throughout the body, venous thrombosis commonly occurs in the deep veins of the legs. Deep veins tend to thrombose, while superficial veins are prone to varicose.

DVT is widely viewed as a complication of hospitalisation (Anderson et al, 1992). It is a multicausal condition due to the additive effects of some risk factors such as acute injury, surgery and some medical illnesses (Rosendaal, 1999). Data extrapolation from four epidemiological studies suggests that the annual frequency of DVT in the general population is approximately 160 per 100,000 (Lindblad, 1988; Lindblad, et al 1991; Anderson et al, 1992 and Nordstrom et al, 1992). DVT is a serious threat to recovery and is the third most common vascular disease, after ischaemic heart disease and stroke (Turpie, 1997; Anands et al, 1998). In the immediate course, DVT is a precursor of over 90% of Pulmonary Embolism (PE), a potentially lethal and acute complication (Borrow & Goldson, 1981). The rate of fatal PE is estimated to be around 60 per 100,000 of the general population (European Consensus Statement, 1991). Wiseman first described PE in 1676 and Homans (1957) confirmed the relationship between DVT and PE. The National Institute of Health (NIH, 1986) lends further support to the close link between the development of DVT and PE.

Randomised clinical trials involving more than 1200 persons indicated that a 68 per cent reduction in DVT was associated with a 49 per cent reduction of PE.

Most deaths due to PE occur suddenly in otherwise healthy patients without any antecedent clinical signs of DVT (Coon, 1976). The Thromboembolic Risk Factors consensus group (THRiFT, 1992) reported that nine per cent of patients admitted to a general hospital die and 10 per cent of these deaths are due to PE.This is approximately 77,000 UK deaths in 1992 as reported by the Office of Health Economics (1996). The Scottish Audit of Surgical Mortality and the National Confidential Enquiry into Peri-Operative Deaths in England (NCEPOD, 1993) have both highlighted the significance and major contribution of PE. It is the most commonly reported cause of maternal death during pregnancy and the puerperium in the UK. (DoH, 1994). Fatal PE is often under-diagnosed as the signs and symptoms prior to death are non-specific and may be similar or attributed to myocardial infarction, pneumonia or other chest pathology (SIGN, 1995). PE is a significant cause of mortality in surgical and non-surgical patients alike. It causes death in over 100,000 patients each year in the United States and contributes to further 100,000 deaths (Dalen et al, 1986). This finding is further supported by the Worcester (Massachusetts) DVT study (Anderson et al, 1992). In this community wide study of 16 acute short stay hospitals in Massachusetts, an annual incidence of 48 per 100,000 DVT and 23 per 100,000 PE were reported. Data extrapolation suggests that approximately 170,000 new cases of venous thromboembolism are treated in short stay hospitals per year in the USA. Sandler & Martin (1989) found evidence to suggest that PE causes 10% of the general hospital deaths. The 5 year retrospective analysis of autopsy reports undertaken by Sandler & Martin provides compelling evidence of the serious underestimation of the true incidence and scope of the problem.

To determine further the frequency of venous thromboembolism, Linblad et al (1991) also studied necropsy reports over 30 years and concluded that the incidence of venous thromboembolism has been remarkably stable and unchanged over the 30 years. During this period the proportion of the population aged over sixty five years has doubled, and this may have masked the benefits of prophylaxis and early mobilisation.

The total number of UK annual deaths from venous thromboembolism has been recently estimated at over 188,900 in the UK (Office for National Statistics, 1995).

Although PE is a leading cause of death in USA and Europe, national morbidity statistics do not always reflect that fact for two reasons. Firstly, the diagnosis is frequently missed and most people, who die as a result of embolism, often have other major medical problems to which death is often attributed (Hirsh &Hull, 1982, Dismuth & Wagner, 1986).

In the longer-term sequelae, patients face an uncertain future as DVT can complicate into Post Phlebitis Syndrome (PPS), a chronic disabling condition (Dalen et al, 1986). PPS occurs in more than 50 per cent of patients (Strandness et al, 1983) and although it is clinically less dramatic in outward clinical presentation, it causes considerable suffering (Kakkar & Lawrence, 1985). There is no readily available data on the prevalence of PPS in UK, but in Sweden and Switzerland 1-2% of the population have Post Phlebitis Syndrome. Approximately 800,000 people in USA have this condition (Dalen et al, 1986). Reportedly, sixty percent of patients with proximal DVT develop post thrombotic syndrome manifested by phlebitis, leg ulceration and pigmentation (Brandjies et al, 1997). Long term complication of PPS commonly results in venous insufficiency and chronic venous leg ulceration. Venous ulcers develop in at least 300 per 100,000 population and the proportion due to DVT is about 25% (Nelzen et al, 1991a; Nelzen 1991b). Nelzen et al, (1991a) claim that there is an underestimation of the leg ulcer problem among the elderly. With an expected increase of elderly population, this problem has to be fully resolved as treatment of venous ulcers costs the NHS about £600 million a year (Drug & Therapeutics Bulletin, 1992).

The long term clinical course of acute DVT is illustrated in table 1.2.

Table 1.2: Long term course of acute DVT

Clinical course	2 years	5 years	8 years
Recurrent DVT	17.5 %	24.6 %	30.3 %
Post thrombotic syndrome	28 %	28 %	29.1%

Adapted from: Prandoni et al, 1996.

Screening studies with objective diagnostic tests, indicate that surgical patients with previous DVT, have a three times higher incidence of DVT following abdominal surgery compared with the same population without a history of DVT (Kakkar et al, 1970).
Nicolaides & Irving (1975) who reported an incidence of 68% also acknowledge the high recurrence rate of DVT in patients with a previous DVT.

DVT: The silent killer

DVT is a hidden condition and a silent killer (Autar, 1996 c; Davis, 1998) and primary prophylaxis is the challenge (Moser, 1989). This can be achieved by early identification of those at risk and the prompt application of the most appropriate intervention, as clinical diagnosis is notoriously unreliable even to the most watchful observer (Sharnoff, 1980). Traditionally, the routine physical examination in patients with suspected DVT included a careful inspection of the leg, measurement of leg circumference and elicitation of Homans'sign.
The typical classic manifestation associated with the diagnosis of DVT is outlined below:

Clinical diagnosis of DVT

- Asymmetrical ankle oedema.
- Increased diameter of one calf, ankle or thigh in relation to each other.
- Loss of concavity of the malleolar space in one leg.
- Heavy and dull aching pain.
- Tenderness over the affected vein in 75% of cases.
- Low grade fever
- Homans'sign positive in less than one third of patients.
- Dilation of superficial veins.

- Mottled and cyanotic skin.
- Phlegmasia cerulea dolens (painful blue inflammation).
- Phlehmasia alba dolens (painful white inflammation).
- Affected extremity is warmer to touch than the other unaffected extremity.

Data: Weinmann & Salzman, 1994; Paiement & Mendelsoln, 1995; Wallis & Autar, 2001.

Barnes (1982) found approximately 50% inaccuracy when using the Homans'sign to diagnose DVT clinically. Homans is the presence of pain in the calf on forceful dorsiflexion, which is considered as a specific sign of distal calf DVT.

When Homans'sign is used diagnostically in symptomatic patients, it is falsely negative in two thirds of cases of DVT and is falsely positive in more than one half of patients suspected of having DVT whose venograms were later found to be negative (Hirsh &Hull, 1982). Typical clinical findings such as leg pain or swelling and shortness of breath or chest pain are absent in more than half the affected individuals, including a majority of patients who die of PE (Goldhaber et al; 1983; Berqgvist & Lindblad, 1985).

Many patients exhibit no signs to indicate the presence of DVT (Ogston, 1987). Objective tests have shown that clinical physical signs are inaccurate with a frequent false positive diagnosis despite careful daily clinical examination (Joffe, 1975; Weinmann & Salzman, 1994). Both DVT and PE manifest few specific symptoms and are clinically insensitive (Clagett et al, 1992).On the other hand, a perivascular tissue inflammation may cause local pain, tenderness and swelling without thrombosis. Furthermore, non-thrombotic obstruction of large proximal veins may cause pretibial and ankle oedema. Such symptoms are therefore very misleading in the diagnosis of DVT (Lambie et al, 1970). Clinical assessment is therefore full of pitfalls and clinical diagnosis of DVT is little better than a flip of a coin (Redman,1988).

Most thrombi are silent when first detected by objective methods, probably because they do not totally obstruct the vein and because of collateral

circulation. DVT is not often suspected until it has propagated or caused PE (Sandler & Martin, 1989).

Even when DVT is symptomatic, the signs and symptoms are non-specific and can mimic those of other conditions affecting the lower limbs (Hull et al, 1981; Prandoni & Mannuci, 1994). Among 87 consecutive patients with clinically suspected DVT, 37 had a musculo-skeletal cause, 12 had impaired lymphatic flow and 4 Popliteal Inflammatory Cyst (Baker's cyst). A diagnosis of DVT suspected on clinical ground is therefore flawed and must be confirmed by a sensitive objective test. Even in high risk patients it has a sensitivity and specificity of less than 50% (Paiement & Mendelsohn, 1996).

The justification for an objective DVT risk assessment arises out of the silent nature of DVT and the unreliability of its clinical diagnosis. As it is difficult to diagnose DVT clinically, its frequency has therefore been underestimated. Additionally, clinical diagnosis is cost ineffective as the majority of patients have their in-patient stay prolonged by 7-10 days (Hull, Hirsh, Sackett,1981). "Minimal leg symptoms may be associated with extensive venous thrombosis, whereas classic symptoms and signs of pain, tenderness and swelling of the leg can be caused by non-thrombotic disorders" (Verstraete, 1997).

A list of the differential diagnosis of DVT is illustrated below (Table 1.3).

Table 1.3: Differential diagnosis of DVT

- Superficial phlebitis
- Muscle strain of calf or thigh muscles
- Cellulitis
- Baker's cyst
- Chronic venous insufficiency
- Nerve compression syndrome
- Lymphodema
- Arterial occlusive disorders.

Data: Verstraete, 1997: p 123.

Monetary cost of DVT and PE

DVT and PE are very costly to the NHS. In 1993, the Office of Health Economic (OHE, 1996) estimated 0.21 million GP consultations for DVT in England and Wales (OHE, 1996). With an average cost of £11.90 per consultation, the cost of DVT in general practice alone is calculated at around £ 2.5 million. Post surgical DVT and PE are estimated to cost the NHS between £204.7 and £222.8 million in 1993 (OHE, 1996). This estimate ignores the costs incurred by the patients and their families. It is estimated that if all the high risk patients (over 45 years and have undergone major surgery) had received adequate prophylaxis, NHS would have made a cost saving of between £33.4 and 81.8 million. The number of deaths due to post-surgical PE would also have been reduced by more than 400. In the United States, the cost of treating a patient for DVT is estimated at $ 2926 and $ 4179 for PE per patient (Oster et al, 1987).

DVT and fatal PE are an easily preventable cause of hospital death (Morrell & Dunnill, 1968; Kendall, 1992).
Essentially, there are two types of venous thromboprophylaxis: primary and secondary. Primary prophylaxis using either pharmacological or mechanical methods or both is directed at preventing the occurrence of DVT. This proactive approach to DVT management requires the identification of those at risk, followed by the administration of the most appropriate thromboprophylaxis. Hull et al (1986) argue that an ideal primary prophylactic approach is one which is free of clinically important side effects and well tolerated and accepted by patients, nurses and medical staff. It should also be easily administered, inexpensive and require minimal monitoring.

On the other hand, secondary prophylaxis is reactive to DVT and is achieved by early detection and treatment of subclinical DVT to prevent PE. Secondary prevention by screening should never replace primary prophylaxis and is reserved for those patients in whom effective primary prophylaxis is either unavailable or contra indicated (Hull, Raskob & Hirsh, 1986).

In monetary terms, primary prophylaxis is considerably less expensive than secondary prevention because the latter requires full anticoagulation of large numbers of patients with subclinical DVT (Kakkar & Stringer, 1990). The average cost of some first level preventative management is less than £10 for patient at risk, on whom at least £ 2,000 is likely anyway, to be spent on the hospital service. The average cost of DVT crisis management is £2,300 (Mackmurdo, 1991). So the first safety decision is whether to spend £2000 on a patient and risk a relatively high number of deaths and a relatively high number of additional £2,300 costs per patient in attempting to treat the crisis. Alternatively, it is prudent to spend £2,010 on a patient and risk a relatively low number of deaths and a relatively low number of additional expenses of £2,300. The expenditure required for preventative measures represents both good patient care and good clinical management (Janssen et al, 1987). Routine DVT prophylaxis could save between 4000-8000 lives annually (Hull et al, 1986).

In the absence of prophylaxis, the frequency of fatal PE ranges from 0.1-0.8 per cent in patients undergoing general elective surgery (Weinmann & Salzman, 1994) and 0.3-0.7 per cent in patients with hip arthroplasty (Kakkar et al, 1997).

Patients in the low risk category, have less than 10 per cent risk of developing calf DVT. Without thromboprophylaxis, DVT incidence ranges from10-40 per cent in the moderate risk group. The reported frequency of DVT varies between 40-80 per cent in the high risk category as summarised in table 1.4

Table 1.4: DVT risk categories

Risk categories	DVT risk	PE risk
Low risk: 0-1 risk factor may be present. Minor surgery with anaesthesia < 30 minutes or Major surgery in < 40 yrs old. No other risk factor is present or Minor trauma or medical illness.	< 5%	0.01%
Moderate risk: 2-4 risk factors may be present. Major surgery in >40 yrs old or Major surgery or lower limb surgery in patients on contraceptive pill. Major medical illness (heart failure or acute myocardial Infarction) with prolonged immobilisation. Leg fracture in a patient < than 40 years old.	5-40%	0.1-1.0%
High risk: > 4 risk factors may be present. Major orthopaedic surgery to lower limbs, including hip or knee arthroplasty or Fractured pelvis, hip, leg. Major surgery in patients with malignancy or Major surgery in patients with previous thromboembolism or > 60 yrs old. Lower limb paralysis or hemiplegia or CVA or Paraplegia Thrombophilia with additional disease Recent history of DVT or PE.	> 40%	1-10 %

Adapted from: National Institutes of Health (NIH, 1986).

Caprini et al, 1991.

Kendall, 1992.

SIGN, 1995

International Consensus Statement,1997

THRIFT 11, 1998.

Although the consensus groups in Europe, United Kingdom and North America each published their own recommendations and therefore differ in some details, their general recommendations are similar.

When no prophylaxis is used, the unnecessary loss of life and the costs of diagnosing and treating established DVT and PE are substantial and the risk unacceptable. Among patients undergoing total joint arthroplasty without

prophylaxis, venous thromboembolic diseases have been reported in 50% to 75% of cases (NIH, 1986). A retrospective venous thromboprophylaxis cost analysis of controlled clinical studies for hip fracture surgery, suggests that prophylaxis does not only decrease post surgical morbidity and mortality but also reduces healthcare expenses for complications of DVT (Mol & Egberts, 1994). Cost analysis factors that are difficult to quantify such as the monetary value of human life, cost of treating PPS and the adverse effects of the prophylaxis itself, were not included in the retrospective analysis. Additionally, in most hospitals, the cost of tests and treatment is not known exactly. Hospitals use reimbursement tariffs that may be different from the real costs. Tariffs also vary among insurance companies. Therefore, the result of any pharmaco-economic study may not be very accurate. Nevertheless, this approach gives a general impression of the total cost of prophylactic measures (Mol & Egberts, 1994). Finally, the clinical management of thromboembolic complications may vary between hospitals and patients.

On the whole, DVT and PE prophylaxis reduces the incidence by 50% or more and Vanek et al (1991) and THRIFT (1992) recommend that prophylaxis should be mandatory in moderate and high-risk patients. OHE (1996) estimates that if all patients at high risk of developing a post-surgical DVT had received adequate prophylaxis, NHS would have made a cost saving of between £33.3 and £81.8 million and the number of deaths due to a post-surgical PE reduced by more than 400. With clinical effectiveness and evidenced-based practice on top of the NHS agenda, much would be achieved by application of those research findings to DVT thromboprophylaxis.

In reality, physicians and hospitals have been very slow to develop consistent policies for preventable conditions. Deaths from PE are so infrequent within the experience of individual medical practitioner that it is tempting not to worry about venous thromboprophylaxis strategies because bleeding from anticoagulated patients is relatively common and worrisome (Becker, 1986).

Clinical negligence and national guidelines

There is a growing concern within the NHS about the rise in the number of claims made against the health authorities for medical negligence and increase in settlement amounts (NAHAT, 1991).

Within a climate of increasing health care litigation, the DoH (1996) seeks to foster strategies and management techniques that will reduce the incidence and the adverse impact of clinical negligence lawsuit. NHS trusts are vigorously advised to reduce incidence and impact by adopting prudent risk management strategies. This means that there should be clear procedure for involving front-line staff, in particular doctors and nurses in undertaking a comprehensive risk assessment.

The Scottish Intercollegiate Guidelines Network in 1995 (SIGN) strongly recommends a national guideline to enable individual clinicians, hospital departments and hospitals to produce local protocols.

A number of controversial issues surround clinical guidelines. It is argued that they are a fetter on clinical discretion, clinical freedom and lead to "cookbook medicine" (Wilson, 1995). Others maintain that they are essential element of safeguarding appropriate health care delivery (NHS Executive, 1996).

Risk assessment should be at the primacy of protocol development and clinical guidelines should take into account include age, past medical history of DVT and the nature of surgery. Explicit guidelines improve clinical practice when introduced in the context of rigorous evaluations (Grimshaw & Russell, 1993).

The aims of the national guideline (SIGN, 1995) are to:

- Identify patients at moderate to high risk of venous thromboembolism.
- Administer and monitor appropriate and effective prophylaxis in such patients routinely.

In the presence of overwhelming evidence supporting the efficacy of venous thromboprophylaxis, it is considered clinical negligence not to provide DVT

prophylaxis to the moderate and high risk patients (Parker-Williams & Vickers, 1991).

Following the development of local protocols, providers can implement and audit their use through the following actions:

- Local protocols should be circulated to all relevant staff and displayed on all units where prophylaxis should be given.
- Patient- specific reminders for staff at time of consultation or admission.
- Continuing education.
- Audit.
- Quality Assurance.
- Funding.
- Research.

Source of data: SIGN, 1995.

Risk assessment: The way forward

Risk management is seen as part of the concept of clinical governance. Under section 18 of the Health Act 1999, there is a statutory obligation for the NHS to promote quality, which requires employer to undertake risk assessment and management (Health Service Circular, 1999).
With a remarkably high incidence of DVT being described as silent, asymptomatic and unsuspected, morbidity and mortality can be reduced by risk assessment followed by the implementation of the most appropriate intervention (Letsky, 1989; THRiFT, 1992).

The challenge for the prevention of DVT is therefore to assess and individualise patient for risk factors, pathophysiology, planned surgical procedures and specific medical condition. An objective and comprehensive risk assessment derived from the known clinical criteria and variables places patients into one of the three main categories: low, moderate and high.

In 1992, a group of UK clinicians (THRiFT, 1992) met in Sheffield and considered the risk to hospital patients and made the following general recommendations:

- All hospital patients should be assessed for clinical risk factors and an overall risk of thromboembolism. Effective prophylaxis mandates a clear understanding of the risk factors in DVT, so that the relevant variables can be controlled.
- Patients should receive prophylaxis according to their risk categories.
- Clinicians, units and hospitals should develop written policies for prophylaxis
- Prophylaxis should be included in the clinical audit and in the patient care plan,

The appropriateness, efficacy and implementation of the different thromboprophylaxis modalities depends on the degree of risk in different patient groups and the nature of the surgical procedures being undertaken. Table 1.5 illustrates the recommended DVT prophylaxis by risk category.

Table 1.5: Venous thromboprophylaxis strategies

Risk category	Recommended DVT prophylaxis
Low risk	Early mobilisation + Active and passive exercises + Graduated compression stockings
Moderate risk	Early mobilisation + Graduated compression stockings + Low dose heparin or Low molecular weight heparin or Intermittent pneumatic compression
High risk	Graduated compression stockings + Adjusted dose of heparin or Low molecular weight heparin + Intermittent pneumatic compression, except for open limb injury.

Data: NIH, 1986
European Consensus Statement, 1991
THRiFT, 1992/1998
International Consensus Statement, 1997

A number of variable based laboratory tests and forbiddingly complex DVT prognostic equations have been developed to predict patients for risk of

DVT (Gallus, 1989). But the lack of standardisation in the laboratory procedures creates inconsistency in the estimated outcomes. This inconsistency and dependence on laboratory data to enable a comprehensive assessment and diagnosis is probably the reason why the prognostic indices have not been widely and successfully applied to date. Laboratory tests tend to lead to a delay and can only be initiated by medical staff and this excludes a significant element of the health care team from contributing to risk reduction.

In the development of a predictive index that is user friendly, reproducible, robust and cheap. Ruckley (1985) suggests that simple criteria such as age, type of surgery, previous DVT, malignancy, obesity and varicose veins could be used to identify those at risk of DVT. Such criteria would fit automatically into the ward routine and are much more likely to be consistently and successfully applied by nurses and doctors as they involve readily available information.

Nursing management of DVT

Nurses are very well placed to make an appropriate DVT risk assessment, followed by appropriate referral to house officers for thromboprophylaxis. The very nature of their duties enables nurses to:

- Provide continuity of care and monitor DVT events.
- Undertake regular ward rounds and assess patient for risk.
- Participate in routine exercise that aid venous return.
- Know patients who are immobile and the nature of the immobility.
- Maintain and administer prophylaxis: primary and secondary.
- Participate in collaborative management of DVT with medical staff.

Safety, effectiveness, simplicity and cost effectiveness are the chief characteristics associated with venous thromboprophylaxis (Caprini et al, 1994). Importantly, nurses take a leading role in the safe administration of effective non-invasive methods of DVT prophylaxis (Reid, 1997). They are able to advocate and apply graduated compression stockings, which increase venous return. Intermittent pneumatic compression devices promote venous return, increase fibrinolytic activity and may be indicated, especially during prolonged surgery. Other non-invasive preventive

measures undertaken by nurses include leg elevation, early ambulation, and exercise for patients who are chair or bedbound, ensuring adequate hydration and avoiding pressure on the popliteal spaces when legs are crossed. Patients who are at risk would receive the appropriate venous thromboprophylaxis by nurses and be observed continuously for signs of DVT such as erythema, unilateral swelling and pain or pressure on the calf muscles (Mills, 1997). Although such clinical observations are often unreliable, they could be used to estimate clinical probability in patients suspected of DVT (Wells, 1995 a). While the design and implementation of protocols currently remain part of prescribed medical care, the incorporation of the nursing process documentation and active involvement of nurses serve to enhance compliance (Byrne et al, 1996). The patient centred approach to nursing emphasises that nurses manage DVT holistically and proactively by minimising risk in contrast to the medical approach that is problem orientated and manages DVT reactively through the crisis intervention model (Autar, 1996 b). A comparative analysis of the patient centred model and the medical model is outlined in table 1.6

Table 1.6: A comparative analysis of the medical and nursing models

Medical model	Nursing model
Doctor- centred	Patient- client centred
Problem oriented	Not problem oriented
Parts of the patient	Whole patient- client
Reductionist approach	Holistic approach
Focuses on what is wrong	Focuses on what is right
Negative approach	Positive approach
Focuses on quantity	Focuses on quality
Treats symptoms	Works with causes
Emphasises secondary prevention	Emphasises primary prevention.

Source: Armentrout, 1993.

Historically, patients with DVT have been treated initially with intravenous unfractionated heparin, necessitating inpatient care for dosing and close monitoring of anticoagulation (Pout et al, 1999).

With the introduction of Low Molecular Weight Heparin (LMWH) and its safe administration and cost effectiveness outperforming Standard Heparin, hospitals are now treating people with DVT in outpatient clinics and at home (Levine et al,1996; Deagle, 1998). Patients are requested to visit the clinic

run by an anticoagulant nurse practitioner. The Southampton University Hospitals Trust (SUHT) who introduced the initiatives has developed a protocol, in conjunction with the Southampton Community Health services NHS Trust, to treat patients with DVT at home, using the community nursing team. Tinzeparin is the LMWH of choice used by SUHT.

This is very cost effective. A medical bed at SUHT costs £350 per night. SUHT had 236 confirmed DVT admissions in 1996/97, with an average stay of 7-8 days and another 400 suspected DVT's who stayed overnight. The cost of the DVT community programme includes 1.5 trained nurse per annum, £74 per patient for drugs and a modest annual payment to the community trust (Deagle, 1998).

To enable them to take over the many functions of the clinic effectively, the consultant haematologists, laboratory staff and pharmacists devise specific training packages. The following are the areas focused on:

- Normal processes of coagulation and the mode of action of Warfarin
- Pharmacology and drugs interaction
- Interpretation of the computer dosing system for anticoagulation
- Laboratory testing and overriding the computer should problems occur
- GPs liaison
- Initiation of anticoagulant treatment
- Maintaining therapeutic control and dosing based on the International Normalised Ratio (INR)
- Managing minor surgical and dental intervention
    - Reversal of coagulation

Source: Brown et al, 1998

The benefits of a nurse-led anticoagulant service are listed below:

Benefits of a nurse-led anticoagulant service

- Elimination of the inconvenience of a week-long hospital stay.
- Avoidance of exposure to hospital infection and risk associated with immobility.
- Autonomy of the anticoagulant nurse for implementation of the patient's entire anticoagulation programme.
- Reduction of bed days and laboratory costs in the local hospital.
- Enhancement of skill base of the nurse-led anticoagulant team.

Source: Bevan et al, 2000.

The role of the nurse in the management of DVT prophylaxis has been further endorsed by a prospective audit undertaken by Byrne et al, 1996. Strategies directed by consultants in placing the burden of responsibility primarily on house officers, to ensure that patients at risk receive the appropriate thromboprophylaxis, achieve only fifty seven per cent uptake. When nursing staff are actively involved in formal risk assessment, venous thromboprophylaxis uptake increases to ninety four per cent. Medical staff do not routinely carry out risk assessment and monitor DVT events; in contrast nurses are well placed to spot such oversights.

Nursing assessment as a domain of practice is undergoing considerable change and refinement. A variety of nursing instruments are now available, enabling nurses to carry out systematic, comprehensive and objective nursing diagnoses. Norton et al (1962) introduced the pressure sore risk scale and Gosnell (1973) and Waterlow (1985) later developed this risk calculator. The Barthel Index (Mahoney & Bartel, 1965) for assessment of the activities of daily living and a number of Pain-o-meters enable nurses to carry out objective nursing assessment to guide practice. The Glasgow Coma Scale (Teasdale, 1975) was developed to enable nurses and doctors equally to monitor patients with an altered level of consciousness so that timely intervention could be initiated.

An investigation into the assessment of clients for risk of DVT confirms inconsistency in the assessment of practice and is based on subjective judgement or standard pre-printed care plan (Autar, 1994). A blanket nursing diagnosis of potential problem of DVT provides only a crude index of measurement of the problem. It fails to address the nature of risk in an individual in the context of multifactoral aetiology.
Unless risk is identified by category and the assessment is tailored to the individual, preventative measures are likely to be inappropriate and therefore ineffective.

Identifying those at risk is equally important as those who are not. It allows for the targeting of the limited resources and ensures that nursing action is individualised and founded on a sound rationale and not ritualistic. A risk assessment system based on clearly defined clinical criteria can yield an index of no, low, moderate and high risk of DVT (Autar, 1998). One risk factor is present in the low risk group. Two to four factors are present in the moderate risk and more than four factors are present in the high risk group (Arcelus et al 1991; Caprini et al, 1991).

The array of assessment strategies available to identify patients at risk of DVT are based on laboratory prediction. The haematological tests are invasive, expensive, time consuming and often unreliable. Estimation of Euglobulin Lysis Time (ELT) is one such specific laboratory test for hypercoagulability. It measures the amount of fibrinolytic activity present: too little leads to intravascular coagulation and too much induces haemorrhagic tendency. To measure ELT, the plasma is diluted and acidified. A precipitate is formed containing plasminogen, plasminogen activator and fibrinogen. The precipitate is then redissolved and fibrinogen clotted with thrombin and the time for lysis on incubation at 37 degrees Celsius is observed. The normal range is between 90 to 249 minutes. As described above, the test is very intricate and fiddly, may be difficult to interpret and requires strict standardisation and precision in the laboratory (Letsky, 1985). On the other hand, fibrinogen measurement is a cheap and widely available laboratory test for hypercoagulability but there is an urgent need to standardise methodology and establish an international standard (Palareti et al, l991).

Test standardisation should reduce the confusion for the reader who encounters widely different mean Fibrinogen levels in different published studies. Haematological tests such as Fibrinogen Degradation Products (FDP) showed promise in the prediction of preoperative DVT (Clayton et al, 1976; Crandon et al, 1980) but the illness for which surgery is performed also influences the preoperative levels of FDP (Lowe et al, 1982). Rheological variables such as plasma viscosity, white cell count and haematocrit values have been applied as predictors for venous thrombosis (Ernst, 1987). Although these blood tests are undertaken routinely, their predictive value in relation to DVT is uncertain (Lowe et al, 1992). D-Dimer measurement is the most recent entrant in this galaxy of haematological tests. D-Dimer is a specific degradation product of crosslinked fibrin that is released when the endogenous fibrinolytic system attacks the fibrin matrix of fresh formed venous thrombi (Moser, 1994). While this new arrival is promising in supporting the diagnosis of suspected DVT (Wells et al, 1995), it is of no value in its primary prevention.

The heavy dependence on laboratory findings also involves delay in processing the investigation. As a result, venous thromboprophylaxis is being withheld until test results become available. For prophylaxis to be effective, it has to be promptly initiated (Caprini et al, 1991). The Department of Health (DoH 1993) has suggested that assessment processes that are overly time consuming and complex should be discarded in favour of prompt intervention and patient-specific approaches. The fatality associated with DVT created the urgency to develop a simple and easy to use instrument that will identify those individuals at risk before a DVT crisis occurs. The Autar DVT risk assessment scale (1994) was developed to fill a void in this area of risk assessment and management and in the spirit of the DoH recommendation. The DVT risk assessment tool is illustrated in figure 1. It is simple, timesaving and can be promptly applied at the time of admission, providing sufficient information for the surgeon or physician who ultimately decides which prophylaxis regimen if any is to be used. Founded on evidence-based risk factors in the genesis of DVT, the DVT scale is designed to have a practical application in diverse clinical areas where DVT is a problem.

Aims of this study

The main aims of the study are to:

1. Evaluate the sensitivity and specificity of the DVT risk assessment scale as a predictive index.
2. Determine the consistency of the DVT risk calculator as an assessment tool.
3. Evaluate the practical application of the DVT scale by registered nurses.
4. Explore the current venous thromboprophylaxis strategies in the context of the consensus groups' recommendations (NIH, 1986; International Consensus Statement, 1997; THRiFT, 1998).

Figure one

Autar DVT Risk Assessment Scale

Original

Name

Unit N°

Ward

AGE SPECIFIC GROUP

Age Group	Score
10 - 30	0
31 - 40	1
41 - 50	2
51 - 60	3
61+	4

BUILD

Body Mass Index (BMI)

wt (Kg) / Ht(M)²

Build	BMI	Score
Underweight	16 - 19	0
Average	20 - 25	1
Overweight	26 - 30	2
Obese	31 - 40	3
Very Obese	41+	4

MOBILITY

Risks	Score
Ambulant	0
Limited (uses aids self)	1
Very Limited (requires help)	2
Chair Bound	3
Complete Bed Rest	4

SPECIAL RISK CATEGORY

Risks		Score
Contraceptive Pill	(20-35 years)	1
	(35+ years)	2
Pregnancy/Puerperium		3

ASSESSMENT PROTOCOL

SCORE < 6 NO RISK

SCORE 7-10 LOW RISK (<10%)

SCORE 11-14 MODERATE RISK (11% - 40%)

SCORE >15 HIGH RISK (>41%)

SCORING

Identify apropriate items, add and record the scores below

Assessor	Date	Score

TRAUMA RISK FACTORS

Score only preoperatively and score only one item in this box

Risk	Score
Head	1
Chest	1
Head & Chest	2
Spinal	2
Pelvic	3
Lower Limb	4

SURGICAL INTERVENTIONS

Minor Surgery <30 Mins	1
Major Surgery	2
Emergency Major Surgery	3
Pelvic	3
Thoracic	3
Abdominal	3
Orthopaedic (below waist)	4
Spinal	4

HIGH RISK DISEASES

Risk	Score
Ulcerative Colitis	1
Anaemia: Sickle cell	2
Polycythaemia	2
Haemolytic	2
Chronic Heart Disease	3
Myocardial Infarction	4
Malignancy	5
Varicose Veins	6
Previous DVT or CVA	7

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Summary

DVT is an invisible and insidious condition that crosses all traditional departments, specialities and sub specialities of clinical practice. It is a threat to recovery and can seriously damage the health of the hospitalised patients. Pulmonary embolism, a potentially lethal and ultimate complication of DVT accounts for 10 per cent of hospital deaths. DVT and PE are preventable and the cost of treating patients with such problems is considerably higher than for primary prevention. Although the treatment of DVT is the province of physicians, nurses have a clear role in its prevention. The justification of risk assessment arises from the silent nature of DVT and its clinical diagnosis being notoriously flawed. The Autar DVT risk assessment scale was developed as a simple and easy to use risk calculator to identify those at risk of DVT as well as those who are not. Placing the patients into one of the risk categories facilitates the application of the most appropriate venous thromboprophylaxis as recommended by the national and international consensus groups.

Chapter Two

This chapter explains the nature of the literature search strategy applied to the study and then focuses fundamentally on the theoretical framework underpinning the development of venous thromboembolic complications. The risk factors in the genesis of DVT and PE are examined in the context of the best external evidence derived from research reports and biosciences.

Literature search strategy

As an essential part of the study, the following two areas of the literature were considered:

- The literature relevant to venous thromboembolic complications, risk assessment scales in general and DVT risk calculators.
- The literature on research methodology and data collection tools for the development, application and evaluation of a DVT risk assessment scale.

The literature on this topic comprised research reports of different materials, including books, articles and national and international consensus group statements on venous thromboprophylaxis. Official and legal publications derived from government statistics in North America, Northern and Central Europe provided vital information on the epidemiology of DVT and the scale of the problem.

In order to provide a focus for the search the following key words were looked at: Deep Vein Thrombosis (DVT)

Pulmonary Embolism (PE)
Venous Thromboembolism
DVT prophylaxis
Risk assessment scales
DVT risk assessment
Clinical risk management
Venous disorders
DVT integrated pathways

The quick reference section of nursing and clinical medical sciences libraries were accessed for specific reference books on venous disorders with particular reference to the universally recognised work of Virchow in the development and causation of DVT. Additional to providing the key words for the search, the quick reference guide offered a better understanding of the natural history and course of DVT, deemed to be important to the content and construct validity of the DVT scale.

The OPAC (Online Publishing Access Catalogue) allowed for the key words defined earlier to be searched by authors, subjects and the Dewey Decimalisation System. The indexes and abstracts were used to search for online databases such as: CINAHL (Cumulative Index for Nursing & Allied Medical Library).

BNI (British Nursing Indexes)

Medline (Medical Literature)

Gateways to the topic were explored on the internet. The following websites were particularly very specific to the chosen subject:

- www. Nelh.nhs.uk/ cochrane.asp
- www. Emedicine,com
- www. Ncbi.nlm.nih.gov/entrez/ PubMed
- www.nelh. Shet.ac.uk/ nelk/ kit/c

The most specific literature on the topic was accessed via the following consensus groups on venous thromboprophylaxis:

- NIH, 1986) National Institute of Health, USA
- European Consensus Statement, 1991
- THRiFT, 1992 &1998 (Thromboembolic Risk Factors Consensus Group
- SIGN, 1995 (Scottish Intercollegiate Guideline Network)
- ACCP, 1996 (Associated College of Chest Physicians)
- International Consensus Statement, 1997 & 2001)

The selection of the material for the study derived from multi sources was primarily determined by the levels and types of evidence attributed to Gray (1997) available on venous thromboembolism (Table 2.1).

Table 2.1: Levels and types of evidence

Level	Type of evidence
1a	Evidence obtained from meta-analysis of randomised controlled trials
1b	Evidence obtained from at least one randomised controlled trial
2a	Evidence obtained from at least one well designed controlled study
2b	Evidence obtained from at least one other type of well designed quasi-experimental study
3	Evidence obtained from well designed non experimental descriptive studies, such as comparative studies, correlation studies and case control studies
4	Evidence obtained from expert committee reports or opinions and / or clinical experiences of respected authorities

Adapted from Gray, 1997.

Some seemingly dated reports (Sevitt & Gallagher, 1961, Gibbs, 1957, Kakkar et al, 1970) have also been considered in this study, on account of achieving the level of evidence defined by Gray (1997) and the pioneering and groundbreaking work of the respected researchers. On the other hand, the publications from the venous thromboprophylaxis consensus groups provided the most up to date literature and compelling evidence based research reports.

Theoretical framework

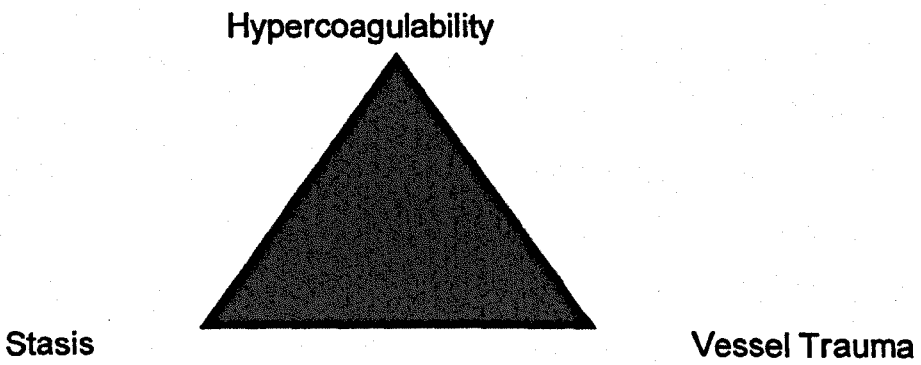
The application of effective venous thromboprophylaxis mandates a clear understanding of the risk factors in DVT, so that the responsible variables can be controlled (Comerata et al, 1989; Clagett et al, 1992). A reduction in morbidity and mortality from DVT can be achieved by timely recognition of the predisposing factors and selective use of prophylaxis (Letsky, 1992). There is now increasing consensus that clinical variables can be used to

predict the high, moderate and low risk groups of patients on admission to hospital (NIH, 1986; THRiFT, 1992; Belcaro, Nicolaides, Veller, 1995).

Our ability to prevent DVT has come with an understanding of its epidemiology, natural history and properly evaluated prophylaxis strategies (Becker, 1986). A fitting tribute is due to Rudolf Virchow (1821-1902) for his pioneering work in providing a comprehensive and sound understanding of the pathophysiology of DVT.

Virchow's classic triad of risk factors in the genesis of DVT is the foundation of our understanding of the cause of DVT and remains valid today (Mammen 1992). The postulated requirements for thrombogenesis, including a triad of factors as first explained by Virchow (1846), are illustrated in figure 2.1.

Figure 2.1: Virchow's triad



The triad of factors for the origin of venous thrombosis are:

1. Vessel Trauma

Changes in the vessel wall due to intimal damage of the endothelium lining the blood vessel. The endothelium is now recognised as not simply being the innermost lining to blood vessels but is also a highly specialised metabolically active interface between blood and the underlying tissue. It maintains vascular tone and predominantly serves to inhibit platelet adherence and initiation of clot formation (Hunt and Jurd, 1998). Conversely, injury to the endothelium exposes highly thrombogenic components to the platelets. Trauma, fracture, thermal injury, surgical

procedures, instrumentation and inflammation may cause injury to the endothelium by endotoxin. Even in the absence of direct trauma to the veins, the widespread vasodilatory effect of many general anaesthetics can result in microtears and disruption of endothelial integrity (Merli and Martinez 1987; Comerata et al, 1989).

2. Stasis

Changes in blood flow due to venous stasis. Immobility is one of the major contributors to venous stasis. Venous stasis causes abnormal blood flow in valve pockets, which are the potential sites for clot formation. Venous stasis prevents clearance of activated coagulating factors and retards inflow of clotting factor inhibitors (Rolling, 1995). Stasis also induces anoxia of the endothelial cells, which release serotonin and histamine, and this provokes local thrombi formation. The endothelial cells contract, exposing the basement membrane and activating the coagulation cascades (Das, 1994).

3. Hypercoagulability

Changes in the composition of blood due to hypercoagulability. Increased platelet activity and or a decrease in physiological anticoagulant and fibrinolytic activity cause increased coagulation. Fibrinolysis is a system, which is concerned with the dissolution of clots as well as the prevention of thrombi formation (Marsh 1981; Poller, 1993). Plasma proteins contain an euglobulin called plasminogen. When a clot is formed, plasminogen is trapped in it. The trapped plasminogen remains inactive until the injured tissue releases tissue plasminogen activator (t-pa) which converts plasminogen into plasmin. Plasmin is a powerful enzyme, which digests fibrin threads and destroys some of the clotting factors.

Increased blood viscosity occurs in response to surgery or injury and this physiological change is known as the "acute phase reaction" (Lowe, 1979), causing "fibrinolytic shutdown" (Stewart-Rose, 1998). Acute phase reaction is a life preserving measure as it is designed to prevent excessive potentially life threatening blood loss. This phase persists for up to about four days (Chakrabarti et al 1969). Oral contraceptives, pregnancy and puerperium are other risk factors contributing to hypercoagulability. Impaired fibrinolysis is noted in obesity and advancing age.

Some common congenital and acquired conditions associated with hypercoagulability are listed below:

- Deficiencies of Antithrombin III
- Protein C deficiency
- Protein S deficiency
- Circulating lupus anticoagulant
- Myoproliferative diseases
- Dysfibrinogenemia
- Disseminated intravascular coagulation
- Malignancy.
- Oestrogen use.
- Inflammatory bowel disease.

Experimentally and clinically, it is now recognised that at least two of these postulated factors in combination are significant in the development of DVT (Mammen, 1992). Virchow's seminal work has made it possible and practical to examine the multifactoral context of identifying those at risk and so allows for the implementation of the most appropriate venous thromboprophylaxis. It is within this conceptual framework that the Autar DVT risk assessment scale was developed and tested.

Risk factors in DVT

A number of clinical circumstances increase the risk of DVT. There is convincing evidence that a number of clinical factors increase the preponderance to DVT (Kakkar et al, 1970; Ruckley, 1985; Caprini et al, 1991). The clinical factors for thromboembolism can be conveniently broken down into factors associated with patients or those associated with the disease or surgical procedure. Patients' factors include increasing age, obesity, varicose veins, immobility, paraplegia, pregnancy and puerperium, previous DVT or PE and congenital or acquired thrombophilia. Factors associated with the disease or surgical procedures include abdomino- pelvic surgery, malignant disease, inflammatory bowel disease, haemolytic anaemia, sickle cell anaemia, polycythaemia and heart diseases. The application of these factors to the patient population forms the basis of identifying the degree of risk (RCOG, 1995). The risk factors for DVT are

associated with vascular injury, activation of blood coagulation and venous stasis, which are the Virchow's triad of factors in the pathogenesis of DVT (Rosendaal, 1997). The thrombosis risk factors that are frequently considered by surgeons when deciding to use prophylaxis are tabled below:

Table 2.2: Risk factors influencing surgeons on when to give prophylaxis

Thrombosis risk factors	Response rate by surgeons (%)
Previous DVT	85
Immobilisation	44
Duration of surgery	43
Hypercoagulability	37
Obesity	32
Malignancy	29
Age	25
Pelvic surgical procedure	22
Varicose veins	10

Data: Caprini et al, 1994; Anderson & Wheeler, 1995.

In this survey, 85% of surgeons consider a previous DVT to be the most important risk factor when prescribing prophylaxis, compared to a response rate of only 10% for varicose veins.

The following well-recognised DVT risk factors will now be critically examined in terms of their potential for causing DVT:

- Surgery
- Trauma
- Increasing age
- Obesity
- Prolonged immobility
- Specific DVT risk factors
- High risk diseases
- The hypercoaguable states.
- Other risk factors which include sex distribution, diabetes, smoking and long distance travel.

Review of the compelling evidence on each of these risk factors will form the basis for selecting the most convincing clinical risk factors for ensuring the construct and content validity of the Autar DVT risk assessment tool.

Surgery

It is well recognised that venous thromboembolism is a common postoperative complication (Lindblad et al, 1991) and is the major DVT risk factor (Moser, 1989).

Nature of surgery:

The nature of surgery carries different weighting risk. Major surgery is defined as a procedure on a patient requiring more than 30 minutes of general anaesthesia and carries higher risk (Goucke, 1989; Moser, 1989). Dilatation of the veins occurs during anaesthesia. Comerata et al (1989) measured venous dilation in patients undergoing hip replacement surgery and found 20 % of patients with the most venous dilation went on to develop DVT. Coleridge-Smith et al (1990) have also demonstrated venous dilation in patients undergoing general surgery. They found that by the end of surgery, the gastrocnemius veins were dilated by 22-28%, providing direct evidence that there are significant increases in the diameter of the deep veins. Incidence of DVT is greater for patients receiving general anaesthesia, being thirty one percentage points higher than for patients receiving regional anaesthesia (Sorenson & Pace, 1992).

In patients undergoing major surgery, the period of risk extends from the time of operation through the first 7-10 postoperative days (Kakkar and Stringer, 1990). The temporal relationship continues to be controversial, but studies reveal that the risk of developing DVT may be present for a period of 3 weeks postoperatively. The onset of DVT occurs commonly after the second postoperative day (Scurr, 1990). Thrombi of clinically significant proportions are present as early as 24-48 hours postoperatively, peak at 5-10 days and may continue for as long as several weeks (Brown and Newman, 1995).

Virchow's triad of risk factors is all present perioperatively, making surgery the front runner in the risk factors in DVT.

Venous stasis:

Stasis is caused by pre and postoperative immobilisation and general anaesthesia with concurrent loss of venous pump function. Thiopentone induction anaesthesia also produces stasis by venodilation. Furthermore, fibrinolytic activity markedly drops with anaesthesia and returns to normal after only two days (Mansfield, 1972). Stasis and surgery combine to set up conditions conducive to clot formation.

Hypercoagulability:

During surgery, thromboplastin is released, increasing coagulability of blood. In addition to decreased clearance of activated clotting factors, a reduction in fibrinolytic activity after surgery may contribute to thromboembolic complications.

Vessel trauma:

Trauma to the vessel wall and increased level of platelet count favour the formation of thrombi. Intraoperative venodilation is associated with focal endothelial damage. As a result, subendothelial collagens are exposed, serving as initiation sites of clot formation (Comerata et al, 1989).

Duration of surgery:

There is a linear relationship between the incidence of DVT and the duration of surgery. Incidence of DVT in surgery less than 2 hours is 20% and 46% for 2-3 hours, escalating to 62.5% in surgical procedure over 3 hours (Borrow & Goldson, 1981). Table 2.3 illustrates the association between incidence of DVT and duration of surgery.

Table 2.3: Association between duration of surgery and incidence of DVT

Duration of surgery	DVT incidence (%)
< 2 hours	20
2-3 hours	46.6
> 3 hours	62.5

Data: Borrow and Goldson, 1981

Types of surgery:

Some surgical procedures are associated with comparatively higher incidence of DVT than others. Table 2.4 outlines the incidence of DVT after different surgical procedures.

Table 2.4: Association between DVT and types of surgery

Type of surgery	DVT Incidence (%)
Knee surgery	75
Hip repair surgery	60
Elective hip surgery	50-55
Urological surgery	9-36
General abdominal surgery	30-35
Gynaecological surgery	25-30
Neurosurgery	20-30
Inguinal hernia repair	10

Data: Das 1994, International Consensus Statement, 1997.

The risk following below waist orthopaedic surgery is greater than for other procedures, partly because of trauma to the tissue surrounding the deep vein and partly due to the difficulty in moving the injured limb to maintain venous return. Manipulation of the leg causes distortion and occlusion of the femoral vein and is claimed to be causative of DVT (Stamatakis et al, 1977).

Trauma

It is generally recognised that DVT and PE are major contributors to late mortality in trauma patients. An even greater contributor to long term morbidity is post-phlebitis syndrome, a long term complication of DVT (Wheeler, 1988; Caroll, 1993). As a result of improved trauma care, large populations of severely injured patients survive for prolonged periods of time in the intensive care setting. Immobilised by multiple fractures or on life support systems, this population is at high risk of DVT and subsequent potentially fatal PE (Kudsk et al, 1989).

The main predisposing factors to DVT and PE are venous stasis, hypercoagulability and intimal vessel damage, which are all present in the trauma patients.

Venous stasis:

Inactivity or bed rest is associated with a decrease in venous blood flow. Besides, in lower limb trauma, due to either the application of plaster of Paris or external fixator, complex wound or traction, the commonly applied DVT prophylaxis such as intermittent pneumatic compression and graduated compression stockings may be contraindicated. With complex leg wounds caused by compound or open fracture, anticoagulation is not always advised as it may interfere with the healing process or increase the risk of haemorrhage. Stasis is particularly important in spinal paralysis patients because it has been shown that venous stasis is markedly increased in the paralysed limb (Swan & Black, 1984). The incidence of DVT is higher in patients with spinal fracture who have complete lesion of the cord being 18% compared to only 8% in those who have incomplete lesion (Watson, 1968).

Venous endothelial damage:

Injury to the venous endothelial lining caused by the trauma, releases thromboplastic substances locally and exposes the basement membrane. This activates the clotting cascade (Shackford & Moser, 1988).

Hypercoagulable state:

Trauma imposes a transient hypercoaguable state because of the reactive effect of circulating tissue thromboplastin and other procoagulants. Additionally, there is a decrease in fibrinolytic activity (Chakrabarti et al, 1969). Further, in states of hypoperfusion, which frequently accompany severe trauma, the liver may not be able to clear the activated clotting factors from the circulation (Hirsh, 1977). Trauma patients also have a moderate increase in catecholamines, which are known to increase platelet aggregation (Hirsh, 1981; Bennett & Towler, 1985).

The reported incidence of DVT in patients with trauma varies between 20 to 90 percent (Shackford, Moser, 1989). There is an increase in the DVT incidence of patients with fractured neck of femur, ranging from 27-83 % (Monterey et al, 1993). However, the inclusion of patients after isolated hip fractures may give an unfair representation of the true incidence of DVT in multiple trauma patients (Kudsk et al, 1989). Patients with hip fractures often

belong to an elderly population with coexisting medical conditions associated with thrombogenesis. In hip trauma, DVT risk starts at the time of injury due to the nature and site of injury (Grace, 1993). Accidental damage to the endothelium occurs during manipulation and repair of the hip (Stamatakis et al, 1977).

Review of literature on trauma patients yielded 8 studies firmly exhibiting variability in the incidence of DVT. The incidence of DVT analysed in the 8 studies is outlined in table 2.5.

Table2.5: Incidence of DVT in trauma patients

Study	Year	No of patients with DVT	DVT (%)
Sevitt & Gallagher	1961	125	81
Freeark et al	1967	124	44
Nylander & Semb	1972	15	7
Silver et al	1975	100	18
Brach et al	1977	10	9
Rossi et al	1980	18	13
Willen et al	1982	38	8
Myllynen et al	1985	37	18

Data from: Autar, 1996 c

Some groups of young patients are also at high risk due to the nature and types of injuries sustained. DVT occurs at a high frequency in patients with spinal cord injury with a reported incidence of up to 72% or more in the absence of prophylaxis (Hull, 1992).

Spine patients have a number of risk factors for developing DVT after surgery: prolonged bedrest, lengthy operative procedure, and manipulation of the great vessels during anterior spine approaches. In addition, nursing the patients in prone position on certain frames may compress the femoral vein (Bret et al, 1993).

Acute spinal cord injury patients reportedly have increased coagulability and decreased venous return (Merli et al, 1988).

The incidence of DVT in spinal injury patients is tabled below: -

Table 2.6: Incidence of DVT in spinal injury patients

Study	No of patients	DVT (%)
Perkish et al,1978	50	16
Rossi et al,1980	18	72
Frisbie & Sasahara,1981	17	6
Myllynen et al,1985	23	100
Merli et al,1988	17	47
Petaja et al,1989	9	67
Yelnik et al,1991	127	23

Data: Hull, 1992

Despite the consensus that DVT and PE are considered common complications after major trauma, the frequency associated with different types of injuries have not been comprehensively quantified. To this end, Geerts et al (1994) undertook a prospective study in a Regional Trauma Unit on 349 patients and reported the wide variability in the incidence of DVT associated with different types of trauma (Table 2.7)

Table 2.7: Association between DVT and types of injury

Injury	DVT (%)
Face, chest or abdomen	50
Major head Injury	54
Pelvic Injury	61
Spinal Injury	62
Tibial Injury	77
Femoral Injury	80

Data: Geerts et al, 1994.

Advancing age

There is a strong correlation between increasing age and DVT development (Anderson et al, 1991; Nordstrom et al, 1992). Post mortem and clinical studies have demonstrated that the frequency of DVT increases exponentially with age (Gibbs, 1957; Rosendaal, 1997). The increased frequency may be due to increased likelihood of coexisting medical condition or surgery. Age represents a definitive risk factor in DVT and is thought to be due to reduced mobility and scarce utilisation of the calf muscle pump with venous stagnation in the veins of the lower limb (Gibbs,

1957). Multiple regression analysis of possible risk factors has also revealed that age is a definite risk factor for DVT (Havig, 1977). As age advances, the calf muscle mass also decreases and the soleal veins increase in number, size and tortuosity (Coon, 1976). Such physiological changes reduce the efficiency of the venous pump and contribute to a decrease in the rate of venous return from the lower limbs.

In ageing, the level of Antithrombin III, which is the direct inhibitor of Factor X, is reduced, leaving the patients more susceptible to blood clotting (Lowe, 1979).

DVT is uncommon in children (Rosendaal, 1999) and rises sharply with age. It is considerably rare in children with a clinical prevalence of 1.2 per 10,000 admissions to a children hospital (Coon, 1976). Even when it occurs it is associated with conditions such as multiple trauma, leg or foot surgery or femoral vein cannulation (Jones & MacIntyre, 1975). No venous thrombosis or embolism was reported under the age of fourteen (OPCS, 1990). The linear relationship between increasing age and DVT is listed under the International Classification of Diseases (ICD) number 453 and illustrated in table 2.8.

Table 2.8: Increasing age and associated venous embolism and thrombosis: ICD 453

Age groups	Recorded cases per 1000
45-49	11
50-54	12
55-59	19
60-64	54
65-69	100
70-74	123
75-79	205

Data: OPCS, 1990: Cause of Mortality Statistics.

The different age groups are another important consideration in the development of DVT. Although it increases sharply with year, risk becomes appreciable after the age of 40 (THRIFT, 1998). In an autopsy survey on a population who died from burns and injuries, isolated venous thrombosis occurred in 47 per cent of patients under 45 years of age, 62 per cent

among patients aged 46- 75 and 74 per cent in those aged over 75 (Sevitt & Gallagher, 1961).

A higher frequency of thrombi has been reported in postoperative patients over the age of 60 (Kakkar et al, 1970) as illustrated in the table below:

Table 2.9: Frequency of DVT in over forties

Age groups	DVT Incidence (%)
40-59	24
>60	45

Data: Kakkar et al, 1970

Samama et al (1993) reported 50 per cent thrombosis in patients between 60-80 years old. The exponential increase was further confirmed by Borrow & Goldson (1981) and Caprini & Natonson (1989) who reported a DVT rate of 20 per cent in 40-60 years old patients having hip surgery. This doubles between the age of 60 years and 70 years and in patients over 70 this figure trebles.

A number of predictive indices have been developed to identify patients preoperatively for risk of DVT and the researchers have all included advancing age as a dominant risk predictor (Clayton et al, 1976; Lowe et al, 1982; Sue-Ling et al, 1986).

Obesity

Obesity, a condition of excess accumulation of body fat, is generally defined using Body Mass Index (BMI). BMI allows for the effects of height on weight and can be calculated using the following equation:

$$BMI = \frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}$$

The BMI is the simplest and most common method of body build categorisation used in clinical practice.

In UK, a BMI of over 30 is commonly used to define obesity in adults. A BMI of between 25 and 30 generally indicates that an individual is overweight.

The term "morbid obesity" may be used when the BMI is greater than 40 (Green, 1997).

The 1991 Health Survey for England reported that 13 per cent of men and 16 per cent of women aged 16-64 had a BMI of more than 30 (White et al, 1993). If these trends continue, in 10 years more than a quarter of adults in UK will be obese, with devastating consequences for health (Jebb, 2000).The obese patients appear to have twice the normal risk of developing post operative DVT than their lean counterparts (Kakkar et al, 1970).

It has been suggested that the risk of DVT in obese patients may be increased because of impaired fibrinolytic activity and prolonged immobility after surgery (Coon, 1984; Poller, 1993). Obesity predisposes to DVT caused by venous stasis: a pendulous abdomen, in particular, mechanically increases intra abdominal pressure and interferes with venous return.

Obesity also causes venous dilation, resulting in venous stasis (Herzog, 1992). Arguably, obesity commonly leads to relative immobility and increases the likelihood of venous stasis. Obesity as a risk factor was further substantiated by Schaub et al (1975). Of the 95 subjects studied, DVT was detected in 52 obese patients as diagnosed by routine I^{125} Fibrinogen scanning.

Whether it is associated with immobility or impaired fibrinolysis, obesity is a risk factor (Becker, 1986). Increased adiposity in women has been confirmed as an important long term factor for significant pulmonary embolism at autopsy (Goldhaber et al, 1983). This Framingham study raises the possibility that weight reduction in obese women may decrease the chances of PE.

Among women matched for age and parity, it was found that women who developed DVT were on average10 lbs (4.5kg) heavier than those who did not (Vessey & Doll, 1969). In women weighing over 76 kg, confidential enquiries into maternal death, reported that one in five women had fatal pulmonary embolism (DoH, 1994). In a post-mortem survey, Coon (1976)

found that 21.9 per cent of those more than 20 per cent overweight had pulmonary emboli compared to 14.4 per cent in the non-obese subjects.

A significant association between obesity and DVT has been reported in some studies (Kakkar et al, 1970, Lowe et al, 1982) but not in others. Nicolaides and Irving (1975) found that the association between obesity and DVT were not associated independently, after controlling for other major risk factors such as age, previous history of DVT and major surgery. In a prospective epidemiological study of 2877 patients undergoing elective surgery, obesity was not found to be a significant risk factor for post operative DVT (Sigel et al, 1974).

In the analysis of variables for a post operative predictive index, obesity as an independent risk factor was ruled out (Sue-Ling et al, 1986). The evidence to justify the inclusion of obesity as a direct risk factor in the causation of DVT is questionable and inconclusive (Ogston, 1987).

Immobility

Immobility as a DVT risk factor is defined in terms of the number of days required before the patient is able to ambulate unaided (Scurr et al, 1987). Venous return to the heart requires the combined efforts of cardiac contraction, respiratory inspiration and expiration, calf muscle pump and the unidirectional leg vein valves. The calf muscle pump is the powerhouse forcing venous blood towards the heart. During quiet standing when the full effect of gravity is exerted, venous pressure at the ankle is approximately 85-90mmHg. Rhythmic contraction of the calf muscle lowers the pressure to less than 30 mmHg (Kaisary, 1980). Lack of movement of the calf muscle causes a drop in venous return and subsequent stasis.

Plasminogen activator is normally synthesised and released from venous endothelium in response to exercise (Vanek et al, 1991) and patients who are immobile have less fibrinolytic activity than active adults (Browse et al, 1977). Immobility causes stasis and is a major contributor of DVT. When normal venous pump function is impaired as a result of confinement in bed rest, venous stasis manifests itself in two ways. First of all, there is an actual decrease in the linear velocity of blood, compromising venous return from

the lower extremities. Secondly, this is followed by dilation of the veins delaying further venous return (Caprini et al, 1988).

There is a striking relationship between the length of confinement to bed and the occurrence of DVT (Gibbs, 1957). In an autopsy series of 253 patients Gibbs (1957) found a strong association between bed rest duration and venous thrombosis; the incidence being only15 percent for confinement to bed for less than a week but more than 80 per cent for more than a week. This association is further confirmed by other studies (Sevitt & Gallagher, 1961; Sigel, 1974). The incidence of DVT associated with bed rest is tabulated below:

Table 2.10: Incidence of DVT associated with bed rest

Study	No of DVT cases	DVT incidence (%)
Gibbs,1957	149	59
Roberts,1963	58	54
Havig,1977	161	62

Early ambulation is generally accepted as increasing venous flow and reducing stasis (Rose 1979). Sharnoff and Rosenburg (1969) compared immobilised patients who have undergone open reduction and internal fixation for fractures with mobile patients with prostatectomies. The conclusion drawn from this experimental study is that immobility is a potent and discriminatory factor in the development of DVT. Immobility as a high risk factor was further supported by Miller et al (1976) who reported a marked reduction in the incidence of DVT in patients with myocardial infarction, who were mobilised within 2-3 days of the onset of their acute attack. Coronary care units now favour the regime of early mobilisation of patients following acute myocardial infarction.

In a comparative study of medical patients with those who have undergone surgery, compelling evidence indicates that immobility due to bed rest has a direct causal relationship whereas surgery *per se* is indirectly responsible (Murray et al, 1970).

In a study of 109 non surgical bedridden patients, Kierkegaard et al (1987) reported a total of 13 per cent incidence of DVT. The study was restricted to

the first 8 days following admission and the investigators claimed that not all events were recorded. A much higher incidence would be reported if the patients were studied for the full duration of their stay in hospital.
The Lassen and Borris (1991) case control study to evaluate the impact of immobility on patients after hip surgery, confirms the importance of early mobilisation in DVT prophylaxis. Incidence of DVT in patients with hip surgery is usually between 48-74 per cent (Madden & Hume 1976). The patients in the experimental group who were mobilised on the fourth day had an incidence of 23 per cent compared to 75 per cent in the control group whose mobilisation was delayed to the ninth day.

The significance of immobility in the pathogenesis of DVT is further substantiated by the following clinical illustrations:

- Spinal cord injury patients with paralysis are at high risk and 10-64 per cent of such subjects develop DVT (Merli et al, 1988).
- A comparison of two small groups of spinal cord injury patients, one with paralysis and the other without, yielded a 100 per cent DVT incidence in the paralysed group but not in the non paralysed patients (Myllynen et al, 1985).
- In acute stroke patients, the paralysed limbs had a 63 per cent rate of DVT as compared to only 7 per cent in the non paralysed limb (Warlow et al, 1976).
- Autopsy studies revealed a relationship between bed rest and DVT (Gibbs, 1957; Sevitt & Gallagher, 1961).
- Pre operative immobility was associated with higher postoperative incidence of DVT (Sigel, 1974).
- Postoperative patients remain at higher risk during the entire period of immobility, especially if they remain immobile after discontinuation of prophylaxis (Gallus et al, 1976).
- Patients with fractures of the lower limbs are at high risk when the limbs are immobilised in a plaster cast (Hamilton et al, 1970).
- Assuming a compromised posture or restricted mobility due to rigid confinement in an air raid shelter or on long confining rides in car or plane and prolonged sitting can cause DVT (Homans, 1954).

In brief, the evidence is compelling and conclusive that immobile patients are a high risk group and that the stasis factor in Virchow's triad is the dominant cause of DVT.

DVT special risk category

Some patients have an additive risk of developing DVT due to specific factors, which predispose to hypercoagulability. For example: patients on oestrogen therapy as a form of contraception and those in pregnancy and the puerperium state belong to a special risk group. Hormone Replacement therapy has also been implicated.

Oral contraceptives

Contraceptive methods are judged by their effectiveness, acceptability and freedom of side effects. Oral contraceptives remain the most popular method of fertility control but are not without side effects. Sartwell et al (1969) and Vessey and Doll's (1970) controlled investigations overwhelmingly support the evidence that oestrogens increase the risk of DVT. One in every 2000 women using oral contraceptives developed DVT compared to one in every 20,000 not using them (Vessey and Doll, 1970). They also reported a DVT occurrence of 2.5 times greater in women aged 35-44 than those aged 20-34. There is an increased risk of DVT associated with the third generation oral contraceptives: progestogens, desogestrel and gestodene (Maling, 1998). Five epidemiological studies have reported the risk of DVT to be twice as high with the third generation oral contraceptives containing desogstrel and gestodene (WHO, 1995). 70-80 per cent of oral contraceptives used in these studies are the "third generation" compared with less than 5 per cent in Australia, 15 per cent in the USA and 50 per cent in UK (Maling, 1998).

From the biological basis of DVT perspective, oral contraceptive therapy causes hypercoagulability due to increased clotting factors, venous stasis as a result of venodilation and reduced blood flow velocity and consequential vessel trauma from increased vascular endothelial permeability (Coon, 1977; BNF, 1994). The general findings from many published trials on the

effects of oral contraceptives, indicate increased in procoagulants, reduction in antithrombin III levels and a depression in fibrinolytic activity (Poller, 1978). Overall, there is an imbalance of the homeostatic mechanism towards hypercoagulability.

Currently, a progressive reduction in the dose of oestrogen to less than 50 micrograms, complemented by better supervision has reduced the risk of DVT (Stadel, 1981). Risk of DVT in women taking oral contraceptives during the month prior to surgery or trauma appears to increase four to six fold (Sartwell & Stolley, 1982). To minimise this risk, it is recommended that women scheduled for elective surgery should stop taking oral contraceptives 3-4 weeks prior to hospitalisation (Guillaband 1985; BNF, 1994). If discontinuation of oral contraceptives is not possible, as in an emergency, and the patients is still on an oestrogen containing pill, heparin prophylaxis and graduated compression stocking should be considered (BNF, 2000). However, opinion remains divided on the management of patients taking oral contraceptive and undergoing surgery. Sue-Ling and Hughes (1988) argue that the routine use of prophylaxis for DVT in women on the pill is probably unnecessary, particularly in those who have no other risk factors. Most women taking oral contraceptives are generally young, slim and fit and they mobilise early after surgery.

Hormone Replacement Therapy (HRT)

There is little doubt that HRT has numerous long term benefits in menopausal women. These include the prevention of osteoporosis and reduction in cardiovascular diseases such as myocardial infarction and stroke. Recently, it has been suggested that HRT has a protective role against Alzheimer's disease (Morris, 1998). The female brain thrives on oestrogen, which controls psychological health. In a way, menopausal women are suffering from natural tranquilliser withdrawal because some of the effects of oestrogen are as therapeutic as the tranquillisers (Stoppard, 2001). HRT also relieves menopause symptoms, especially hot flushes, night sweats, tiredness and irritability (BNF 2000).

However, HRT has been linked with the occurrence of thromboembolism (THRIFT, 1998). At the time of the literature review and at the launch of this study, there was no available data to indicate whether or not HRT increases

the risk of venous thromboembolism. No causal relationship between postmenopausal oestrogen replacement therapy and DVT has been identified (Gow & MacGilluray, 1971; Boston Collaborative Drug Surveillance, 1974; Moore, 1976). It must be noted that most women who are receiving HRT may have other risk factors, particularly age or undergoing gynaecological surgery. Notelovitz & Ware (1982) and Carter (1992) have also found no association between HRT and DVT.

At the time of the development, application and evaluation of the Autar DVT risk assessment scale (1994), available data were insufficient and inconclusive (RCOG, 1995) and HRT was not considered as a risk factor in the DVT calculator.

On the other hand, two recent studies (Daly et al, 1996 and Jick et al (1996) have both demonstrated that current use of HRT is associated with an increased relative risk of 3.5 and 3.6 respectively of developing DVT. Grodstein et al (1996) reported that the risk of PE was about twice that in non users but this finding was based on only five cases of current users of oral contraceptives together with HRT. To evaluate further the association between HRT and risk of venous thromboembolism, Perez Gutthann et al (1997) conducted a case control cohort study of 347, 253 women aged 50 to 79 without major risk factors for DVT. It confirmed previous findings and concluded that current users of HRT have an overall twofold increased relative risk of venous thromboembolic diseases.

Although HRT confers some health benefits, in several observational and one randomised controlled trial study, Lowe et al (2000) reported a threefold relative risk increase of venous thromboembolism.
In the light of these recent findings supporting the association between HRT and DVT; hormone replacement therapy as a risk factor will be considered in the revalidation of the Autar DVT scale.

Pregnancy and puerperium

Normal pregnancy brings about homeostatic changes. An increase in coagulation factors such as raised fibrinogen, platelets and suppressed fibrinolysis serve to combat the danger of haemorrhage at delivery. Plasma

fibrinolytic activity is decreased during pregnancy, remains low during labour and delivery and returns to normal within one hour of placental delivery (Bonner et al, 1969). As a result, pregnancy is associated with a high risk of venous thromboembolism, accounting for the highest cause of direct maternal death (DoH, 1996). The direct causes of maternal deaths in UK between 1991 and 1993 are broken down in Table 2.11.

Table 2.11: Direct causes of maternal deaths in UK (1991-1993)

Direct causes	Frequency (%)
Venous thromboembolism	27.1
Hypertensive disorders	15.5
Early pregnancy	14.2
Haemorrhage	11.6
Amniotic fluid embolism	7.8
Other direct deaths	7.5
Sepsis	7.0
Anaesthesia	6.2
Genital tract trauma	3.1

Data: DoH, 1996.

Like general obesity, a pendulous abdomen in pregnancy increases intra-abdominal pressure which delays venous return. In addition, during pregnancy as the gravid uterus grows heavier, increased pressure on the pelvic veins reduces blood flow, especially venous return from the legs. Bedrest, immobility and obesity may exacerbate this state of venous stagnation (Allotey & Louca, 1997).

Thrombotic risks in pregnancy can be divided into those of the antepartum and the puerperium period. Antepartum occurs before the onset of birth. Puerperium covers the period immediately following delivery until the reproductive organs return to their non pregnant state. It usually lasts for 6-8 weeks. Antepartum risks reflect the physiological changes of pregnancy such as changes in venous stasis in the lower limbs. Puerperal risks reflect tissue breakdown plus an additional traumatic or surgical component such as prolonged labour or caesarean section. It is generally recognised that caesarean section delivery has a higher maternal mortality and morbidity due to venous thromboembolic diseases. The report on the Confidential Enquiry into Maternal Deaths stated that 13 of the 17 maternal deaths were

from thromboembolism in the puerperium (DoH, 1996) during the three years.

The incidence of DVT is consistently higher in the puerperal period than the antepartum period. In a study of the incidence of DVT among a group of 14,869 pregnant women, Kierkegaard (1983) found a 0.13 per 1000 incidence of DVT in antepartum and 0.16 per 1000 in the puerperium attributable to increased blood viscosity. Two physiological changes increase viscosity in the puerperium. In the early post partum days, there is a major diuresis as the kidneys excrete the unwanted body fluid. The blood becomes more coagulable due to liberation of clotting factors to prevent post partum haemorrhage and decreased in the fluidity of plasma from the diuresis (Beischer & Mackey, 1986).

The relative risk of DVT and PE in women who are pregnant or post partum is 5.5 times greater than in non pregnant and non puerperal women not taking oral contraceptives (Coon, 1976). The prevalence has been estimated to be 2.5 per 1000 deliveries (Bonner, 1981). In the triennium 1976-1978, 47 UK deaths were coded to PE following DVT, with one third occurring in the antenatal period (DOH, 1982). In the triennium 1988-1990, 33 maternal deaths from pulmonary embolism were coded for venous thrombosis (DoH, 1994). Predisposing factors such as gross overweight, increasing age and operative form of delivery augment the risk of DVT (Letsky, 1985). A decline in mortality from pulmonary embolism after a DVT could be due to trend towards young mothers and smaller families, the reduction in traumatic operative delivery and early mobilisation.

High risk diseases

Data documenting DVT in medical patients are less extensive than those of the surgical patients. A number of medical conditions are associated with an increased risk of DVT and PE (Arcelus et al, 1991). Such clinical conditions are:

- Ulcerative colitis
- Hypercoagulable states such as sickle cell anaemia, haemolytic anaemia and polycythaemia.
- Cardiac conditions such as heart failure and myocardial infarction

- Malignancy
- Varicose veins
- Cerebrovascular accident
- Previous DVT.

Ulcerative colitis

Inflammatory Bowel Disease (IBD) patients have a threefold increased risk of developing DVT and PE (Bernstein et al, 2001). Ulcerative colitis is a chronic inflammatory bowel disease, in which part or whole of the large bowel becomes diffusely inflamed with haemorrhagic tendencies. Edwards and Truelove (1964) first documented the association between DVT and chronic ulcerative colitis. Overall, a low incidence of 1.3 to 6.4 per cent has been reported in inflammatory bowel disease. During a 10-year period from 1970 to 1980, thromboembolic complications developed in 92 out of the 7,199 patients (1.3%) with chronic ulcerative colitis (Talbot et al, 1986). The occurrence of DVT in patients with ulcerative colitis appears predictable since it is associated with many of the factors known to encourage thrombi formation: anaemia, dehydration, prolonged bed rest and surgical procedures (Graf et al, 1966). Ulcerative colitis is not necessarily an independent risk factor for thromboembolic complications. Most DVT is reported during the acute phase of the illness (Lam et al, 1975) when other risk factors are also present.

Ulcerative colitis may be a secondary hypercoaguable state. Drug interaction may also result in ineffective anticoagulation and recurrent thrombosis (Deykin, 1970). Prednisolone, which is often prescribed to counteract the inflammatory bowel condition, is also reported to decrease fibrinolysis by inhibiting plasminogen activator production (Lang, 1983).

Physiological changes such as an increase in clotting factor VIII and fibrinogen concentration occur in response to the haemorrhagic tendency. Ulcerative colitis is associated with hypercoagulability and raised platelet count, favouring thrombi formation (Wyshock et al, 1988).

Haematological conditions

DVT is often a complication of sickle cell anaemia and haemolytic anaemia. They are characterised by increase in blood viscosity, significantly raised platelet count and venous stasis, creating an internal environment, which favours the formation of thrombi (Serjeant, 1992). However, the overall incidence of DVT is low (Bell et al, 1977).

In patients with polycythaemia vera, an elevated haematocrit value and increased blood viscosity clearly play a role in the pathogenesis of thrombotic complications. Pearson and Wetherley-Mein (1978) have demonstrated a positive correlation between the incidence of vascular occlusive episodes and the haematocrit level in patients undergoing treatment for polycythaemia.The Polycythaemia Vera Study (PVS) group has also reported a markedly increased risk of DVT in patients over 70 year old, particularly those with a prior thrombotic event (Wassermann et al, 1981).

Blood groups

Blood group A has been implicated as a risk factor. Patients with O blood group have been reported to have half the incidence of DVT compared to those in group A. Jick et al (1969) found that O group is under represented with venous thrombosis compared to other groups. They also found higher level of antihaemophilic globulin in the plasma of A compared with O patients.

Mourant et al (1971) collected and analysed data from 17 studies in connection with blood groups and DVT. They claim that group A has a tendency to thrombose while the O group bleed. However, due to the pattern of the ABO blood group distribution in the population, the sample in those studies was too small to draw any firm statistical conclusions. The relative proportions of blood groups vary from country to country. In UK, 47 % of people are group O, 42% group A, 8% group B and 3% group AB (Campbell, 1993). Nordstrom et al (1992) also found no significant difference in the incidence of DVT for blood group A, B, AB and O.

Cardiac conditions

Clinical and pathological studies report a relationship between certain cardiac conditions and venous thromboembolism. Heart diseases are associated with cardiac arrythmias, elevated venous pressure and immobility, which result in venous stasis. The frequency of DVT and PE is three and a half times greater in patients with heart diseases than those without (Coon, 1976). Patients with atrial fibrillation and congestive heart failure are particularly at risk and an incidence of 10-20 per cent has been reported. In patients with acute myocardial infarction DVT incidence ranges between 20 to 40 per cent (Carter et al, 1987). Claggett et al (1992) reported an overall incidence of 24 per cent among myocardial infarction patients not receiving antithrombotic therapy (Table 2.12).

Table 2.12: Incidence of DVT in patients with myocardial infarction with and without prophylaxis

Regimen	No of trials	No of patients	No of DVT	Incidence (%)
Control subjects	4	214	51	24
Low dose heparin	4	165	11	7
High dose heparin	2	70	3	4

Source of data: Clagetts et al, 1992.

Simmons et al (1973) studied 98 patients admitted to a coronary care unit following acute myocardial infarction and reported a DVT incidence of 27 per cent. The study also confirms that DVT is more likely to develop in patients over the age of 60 and with a past medical history of angina pectoris, left ventricular failure and congestive cardiac failure.

Maurer, Wray and Shillingford (1971) reported a 37 per cent incidence of calf vein thrombosis among the 90 patients admitted to a coronary care unit who were not anticoagulated. In a preliminary study, for patients with confirmed myocardial infarction not treated with anticoagulants, the incidence of DVT was 38 per cent and those treated was 5.5 per cent. This preliminary study supports the relatively high incidence of DVT also reported by Maurer et al, (1971).

In a series of descriptive studies using leg scan positivity to diagnose DVT in patients who had myocardial infarction, an incidence of 20-40 per cent has

been recorded (Carter et al, 1987). However, myocardial infarction was not proven to be an independent risk factor since the treatment of the patients in these studies involved prolonged bed rest. Many other factors are also associated with myocardial infarction. Advanced age, a history of thromboembolism and the presence of varicose veins may augment the risk of DVT. In fact, most studies related to patients with myocardial infarction are old dating from a time when a different mobilisation routine was dominant (Bergqvist, 1988).

Malignancy

The relationship between neoplastic diseases and DVT has long been recognised, since Armand Trousseau (1865) first reported a high incidence of DVT in a series of patients with gastric carcinoma. In the majority of cases, DVT occurs after the clinical detection of cancer (Goldberg et al, 1987).

The overall incidence of DVT in patients with cancer has been reported to be between 1 per cent and 11 per cent but in post mortem studies this is considerably higher (Rickles & Edwards, 1983). Post mortem studies by Sproul (1938) confirmed the high frequency of DVT in patients who had died of cancer. In fact, cancer is often seen in patients with DVT. It is five times more commonly diagnosed within six months after DVT than a controlled group matched for age and sex (Nordstrom et al, 1994). Ahmed and Mohyuddin (1996) also found 3 cases of cancer in 113 patients with primary DVT (2.65%).

In postoperative patients, the presence of cancer increases the risk of DVT threefold when compared to patients without cancer, regardless of other risk factors (Kakkar et al, 1970). Browse et al (1977) reported an incidence of 56 per cent DVT in patients with malignancy and this figure is similar to those of other previous studies (Kakkar et al, 1970).

Surgical procedures also increase the risk of DVT in patients with cancer to a greater extent than in patients with non malignant conditions. Pineo et al (1974) reported 10 of the 30 patients with cancer developed DVT following

abdominothoracic surgery as opposed to only 14 of 134 control subjects undergoing similar procedure for non malignant conditions.

An increase of DVT associated with cancer can possibly be explained on the basis of prolonged surgery and extended post operative bed rest (Gallus, 1976; Hirsh et al, 1981). In addition, malignant tumours are often associated with advanced age, which may enhance the risk of DVT.

Malignancy is associated with hypercoagulability. Abnormalities in routine blood coagulation tests have been reported to occur in as many as 92 per cent of patients with cancer. The most common clotting abnormalities are elevated levels of fibrin and fibrin degradation product (Rickles & Edwards, 1983). Patients with malignant disease and those with thrombosis have a fibrinolytic shut down favouring thrombi formation (Browse et al, 1977). Because malignancy commonly is associated with a number of other risk factors, the direct effect of malignancy is uncertain.

Varicose veins

Anatomically, veins have semilunar valves, occurring at regular intervals. The valves are arranged so that blood flow is towards the heart (venous return). Stretching the veins increases their cross-sectional areas, but the valves do not increase in size. As a result, the valves do not close completely. When this develops, the pressure in the veins of the leg increases still more owing to failure of the venous pump. Consequently, venous stasis is inevitable. Varicosities of the veins cause phlebitis, leading to increased risk of DVT. Varicose veins are associated with venous stasis in the pathogenesis of DVT.

Varicose veins as a risk factor for DVT have received considerable publicity by both the national and international consensus groups (NIH, 1986; International Consensus Statement, 1997; THRiFT, 1998). Schaub et al (1975), who reported a 48 per cent incidence of DVT, found that more than half of the patients in the study had a history of varicose veins.

The following studies have all included varicose veins as a risk factor in their predictive equation for DVT:

- Kakkar et al, 1970
- Nicolaides et al, 1975
- Clayton et al, 1976
- Lowe et a, 1982
- Sue-Ling et al, 1986

However, opinion remains divided as to whether varicose vein is an independent risk factor. Although Kakkar et al (1970), Nicolaides et al (1971) and Coon (1977) claim that varicose vein is a powerful discriminatory DVT risk factor, its significance as an independent factor remains controversial. May (1979) reported only one case of DVT in a study of 30,000 patients undergoing surgery for removal of varicose veins. On the other hand, varicose veins may coexist incidentally with other well-documented risk factors to cause DVT. For example, occurrence of DVT is positively associated with history of varicose vein in patients taking HRT (Perez Gutthann et, 1997). Gallus (1976) also concedes that varicose vein may contribute to the risk of DVT but concludes that definitive work in this area has still to be conducted. While general surgeons recognise varicose veins as an associative risk factor, necessitating some form of venous thromboprophylaxis, vascular surgeons are sceptical about this association. In a survey of DVT prophylaxis for varicose vein surgery only 29 percent prescribe venous thromboprophylaxis (Campbell and Ridler, 1995).

Cerbro-Vascular Accident (CVA)

Venous thromboembolism is a major and often unrecognised cause of morbidity and mortality in patients after acute CVA (Strokes) (McCarthy et al, 1977).

DVT of the lower extremities is reported to occur frequently in patients who have had a stroke (Warlow et al, 1972; Warlow et al, 1976). Within a few days after a stroke, 40- 53 per cent of patients develop DVT as diagnosed with I^{125} fibrinogen technique (Warlow, 1978). However, the fact that I^{125} fibrinogen technique does not detect thrombi in the upper thigh suggests that the incidence of DVT was underestimated.

The DVT occurs exclusively in the paralysed leg and this finding is consistent with 42 chronic hemiplegic patients where the frequency is also 42 per cent (Kamal, 1987). The incidence of DVT in stroke patients has been reported as high as 75 per cent (Brunner & Suddarth, 1992).

Physiologically, changes in haemostatic parameters occur after stroke. There is a rise in plasma fibrinogen and an increase in fibrinogen and fibrin formation. An increase in platelet count contributes to blood hypercoagulability. Prolonged immobility whether due to lack of power or sensory loss may apply pressure on the calf and initiate venous thrombosis (Warlow, 1978).
Without prophylaxis, DVT occurs in 60-70 per cent of patients with dense hemiplegia and between 1-2 per cent suffer fatal PE (Turpie et al, 1987). Sioson et al (1988) also found that 33 per cent of stroke patients admitted to a rehabilitation hospital developed DVT.

Previous DVT

A clinical history of previous DVT has been shown to be strongly associated with an increased frequency of venous thrombosis (Carter et al, 1987). In one of the studies, stepwise logistic regression of the clinical variables has indicated that a previous history of DVT is an independent risk factor (Nicolaides & Irving, 1975). All the three elements of Virchow's triad are embraced, explaining why recurrence of the condition is so remarkably high. Physiologically, venous thrombosis causes scarring of the intimal lining of the vein and a nidus for future clot formation (Hirsh et al, 1981). Secondly, valvular destruction following the DVT impairs the venous pump, thus causing venous stagnation in the lower limbs (Christopoulos et al, 1989). Thirdly, there is a depression in the fibrinolytic activity in patients with post thrombotic syndrome compared to patients within the normal population (Poller, 1993).

Screening studies with objective diagnostic tests indicate that surgical patients with a past history of DVT have a threefold incidence of DVT following abdominal surgery compared with a population without a history of

DVT (Kakkar et al, 1970). They reported an incidence of DVT as high as 68 per cent.

Coon & Willis (1973) studied the recurrence of venous thromboembolism and found that the risk was very high immediately after hospital discharge and then gradually declined to reach a plateau after a period of three years.

In a retrospective analysis, Badaracco & Vessey (1974) estimated that there was about a 12 per cent risk of DVT or PE in pregnancy if a woman had a history of thromboembolism. The risk was not affected by the circumstances of the previous event, whether associated with pregnancy or oral contraception.

Nicolaides & Irving (1975) reported a frequency of 61 per cent in patients with previous DVT, compared to 26 per cent in patients without a prior history. Dalen et al (1986) claim that patients with previous DVT when exposed to stressful situations such as surgery, are four times more at risk of developing new DVT. Based on available findings, Rocha et al (1988) and Caprini et al (1991) utilised previous DVT as a powerful predictive variable in their prognostic indices. A previous DVT/PE (appendix 1) is assigned a risk factor value of 3 (Arcelus et al, 1991; Caprini et al, 1991). In Nordstrom et al's study (1992) a past history of DVT was found in 26 per cent of cases and is considered to be a major risk factor for the development of recurrent thromboembolism.

The long term course of acute DVT is illustrated in table 1.2. Patients with DVT have a high risk of recurrence of the condition that persists for many years (Prandoni et al, 1996). The cumulative incidence of recurrent DVT is 17.5% after 2 years, rising to 24.6% after 5 years and 30.3% after 8 years. In the SIRIUS study, Samama et al (1993) reported that a past history of DVT or PE has an odds ratio of 7.9 for the development of a recurrence during hospitalisation. Thus, a patient with previous DVT is nearly eight times more likely to develop a new episode during a subsequent period of increased risk, as compared to an otherwise similar patient without a previous history of DVT or PE.

The Hypercoaguable states

Patients with hypercoaguable states have laboratory abnormalities or clinical conditions that are associated with an increased risk of thrombosis. This category also includes patients with recurrent DVT with no recognizable predisposing factors (Shafer, 1985). The number of hypercoaguable states is still growing but the nine that have been confirmed and classified as Thrombophilia or Hereditary Thrombotic Diseases (HTD) are listed below:

- Protein C deficiency
- Protein S deficiency
- Antithrombin III deficiency
- Plasminogen deficiency
- Dysfibrinogenia
- Tissue Plasminogen Activator deficiency (t-PA).
- Plasminogen Activator Inhibitor excess (PAI).
- Heparin cofactor II
- Factor V Leiden

Of these hypercoaguable states listed above, Protein C (10%), Protein S (12%) and Antithrombin III deficiencies (3%) are most common (Cooper, 1994). Factor V Leiden is an inherited mutation in the gene coding for Factor V and accounts for about 5% of the white population. It causes activated protein C resistance (APCR) resulting in an increased susceptibility to develop DVT (Vandenbroucke et al, 1996).
About 70 per cent of cases of hereditary thrombotic disease have no known genetic cause (Wood & Bunn, 1994).

Protein C and Protein S deficiency are inherited as an autosomal dominant trait and are both vitamin K-dependent proteins similar to factors VII, IX, and X and prothrombin (Marlar & Mastovich, 1990).
Patients with decreased levels of Protein C and S are at high risk of venous thromboembolism and life long management with warfarin is recommended for this patient population (Griffin et al, 1981; Broekmans et al, 1983).

Antithrombin III is a single chain plasma glycoprotein. Through its inhibition of thrombin, and other activated coagulation factors, Antithrombin III plays a central role in the regulation of haemostasis. Deficiency of Antithrombin III is

responsible for two per cent of thromboembolic diseases (Rosenberg, 1975). 90-95 per cent of patients with thrombophilia develop DVT (Marlar & Mastovich, 1990). However, this DVT occurs in specific vessels (mesenteric, spleenic, hepatic) and based on its hereditary nature is usually recognised by surgeon and neurologist. The thrombotic state may occur spontaneously or related to some other underlying hypercoaguable risk factor such as trauma or pregnancy.

Hypercoaguable states are resistant to conventional antithrombotic therapy (Shafer, 1985). Most patients are treated indefinitely with warfarin because of their predisposition to recurrent thrombotic events (Merli & Martinez, 1987).

The clinical indicators of primary hypercoaguable state as described by Shafer (1985) are summarised below:

- Family history of thrombosis
- Recurrent thrombosis without any apparent risk factor
- Thrombosis in unusual sites
- Thrombosis at an early age
- Resistance to conventional venous thromboprophylaxis. Confirming a diagnosis frequently means life-long treatment with anticoagulants.

Other miscellaneous risk factors

A number of miscellaneous risk factors have been frequently cited in the development of DVT. Smoking, diabetes and gender have been implicated and recently long haul travel thrombosis has received much publicity. Each of the risk factors will now be considered in terms of their association and potential for DVT causation.

Smoking

The association between smoking and cardiovascular disorders such as coronary heart disease and myocardial infarction is well-documented (Meade, 1983; Connaughton, 2001). The citation of smoking as a possible risk factor for DVT causation comes as no surprise. It is assumed that

smoking increases platelet adhesiveness by the release of catecholamines such as epinephrine caused by nicotine absorption. An alternative explanation is that smoking increases platelet adhesiveness by release of fatty acids, which may increase the build up of fibrinogen (Howel, 1970).

Vessey & Doll (1969) first reported the association between cigarette smoking and DVT. In the study of the relationship between oral contraceptive and DVT, Vessey and Doll (1969) noted that their affected patients were heavier smokers than the controls. However, the difference between the case and control group was not statistically significant. In the study of mortality in relation to smoking among British doctors, no association was found between mortality from DVT and smoking (Doll & Peto, 1976).

Analysis of cigarette smoking as a clinical variable for DVT demonstrates a negative association (Clayton et al, 1976). This report is further supported by Lowe et al (1982) who concluded that cigarette smoking is a subjective clinical variable and not a predictive index for DVT. Samama et al (1993) did not find any association between DVT and smoking in medical patients.

On the other hand, increasing levels of cigarette smoking were found to be associated with a reduced incidence of DVT. Handley and Tether(1974) found that DVT incidence in the patients who gave a history of regular cigarette smoking within the month before admission was significantly lower than that of non smokers. This finding is confirmed by Marks and Emerson (1977) who reported leg thrombosis in 50 per cent in the control group who did not smoke but only 2 per cent in those who were cigarette smokers. The protective effect of cigarette smoking is observed in all ages in both medical and surgical patients (Prescott et al, 1978). Table 2.13 illustrates the incidence of DVT in relation to smoking habits.

Table2.13: DVT incidence and smoking habits

Smoking habit	Number of patients screened	Number with DVT	DVT (%)
Non smoker	219	43	20
Pipe smoker	21	4	17
Cigar smoker	3	0	0
1-9 cigarettes daily	50	7	14
10-19 cigarettes daily	92	9	10
20-39 cigarettes daily	89	9	10
40+ cigarettes daily	12	0	0

Data: Prescott et al, 1978.

Overall, there is sufficient evidence to conclude that smoking is not a risk factor. In some circumstances, smoking may even have a protective effect against DVT as seen in some patients after myocardial infarction: the smokers in the group had a lower incidence of DVT than the non-smokers (Meade, 1983).

Diabetes as a risk factor

Diabetes mellitus is the most common endocrine disorder known to diffusely involve the vascular system, predominantly the arterial side (Millbank, 1970). Although diabetes has frequently been cited as a risk factor for DVT, it is not certain whether patients with diabetes mellitus are at greater risk than non diabetic subjects. Relatively little literature exists on the question of venous thrombosis from diabetes *per se*.

A frequency of 20 per cent of PE was reported in 349 diabetics aged 30 or over (Coon, 1976). However, of the 71 patients with PE, 52 had associated heart disease and 12 had other risk factors for DVT. In an autopsy study with multiple regression analysis of risk factors, Havig (1977) found no association between diabetes and PE. Diabetics are susceptible to disease of the large arteries, especially those supplying the myocardium and the microvessels of the retina (Banga & Sixma, 1986). Arterial diseases are due to atherosclerosis or arteriosclerosis while venous diseases are the result of endothelial or valve damage (Rosendaal, 1997).

Disturbance of fibrinolytic activity has been implicated in diabetic patients. Decreased fibrinolytic activity has been detected in diabetes, particularly in Non Insulin Dependent Diabetes Mellitus (NIDDM). However, the relationship of these changes to vascular complications needs to be clarified by further studies (Banga & Sixma, 1986).

Sex distribution

Although gender difference has also been mentioned as a risk factor the percentage of males and females with detectable PE at autopsy is almost identical (Coon et al, 1973). In the Worcester DVT study, Anderson et al (1991) reported a higher incidence of DVT for female subjects. However, after taking into account the fact that women live longer than men and risk factors such as pregnancy, the puerperium and the use of oral contraceptives, it appears unlikely that sex difference exists in the incidence of DVT. In hospital, men and women are reported to be equally affected (Coon, 1976).The incidence of acute DVT in the population of Malmo, Sweden, during 1987 revealed no significant difference between men and women (Nordstrom et al, 1992).

Seasonal variations as a risk factor

In many temperate countries, coronary events and strokes are more common in winter than summer (Wilshurst, 1994). Fatal PE has also been reported more often in winter (Gallerani et al, 1992).

Physiologically, this trend is explained in terms of changes in coagulation factors and peripheral vasoconstriction, leading to reduced blood flow in the legs. On the other hand, venous stasis due to venodilation is greatest in summer (Bournemeaux et al, 1996). To test these uncertainties, Bournemeaux et al (1996) reviewed data on patients with DVT over five years. They concluded that in Geneva, a city with a continental climate with temperature differences between summer and winter, there is no seasonal variation in the incidence of DVT.

Bouley et al (2001) analysed discharge data for DVT and PE in France over four years and claim that a clear seasonal variation exist in hospital admission for DVT and PE, suggesting thrombogenic factors could involve a seasonal trend. A sedentary lifestyle in winter encourages reduced mobility and predisposes to venous stasis. However, the study was retrospective and the accuracy of diagnosis of DVT and PE might be questioned.

Traveller's thrombosis: myth or reality

Traveller's thrombosis refers to a thromboembolic disease occurring generally after a prolonged airflight. DVT may be apparent a week or two after the flight and may not be preceded by a painful calf (Cruickshank et al, 1988). The term "Economy Flight Syndrome" was coined by Cruickshank et al (1988) who reported three cases of venous thrombosis after flying. Economy Class Syndrome raises the notion that the degree of being cramped may be an important aetiological factor. However it should be noted that one of the three cases travelled by business class and the other two cases were doctors. On this evidence, it could equally be called travelling doctor's syndrome. There is plausible physiological explanation as to why sitting for long periods might predispose to DVT (Milne, 1992). It seems likely that cramped seating for prolonged periods puts pressure on the popliteal and femoral veins and may have led to clot formation (Homans, 1954). The development of DVT or PE is not limited to air travel. Homans (1954) described a 19-year-old woman with DVT after a drive from Nebraska to Boston. A very important factor in DVT and PE is likely to be the effect of immobility (Moyses, 1988).

During flight dehydration occurs, and this can be further exacerbated by alcohol consumption, causing venous stasis and predisposing to DVT (Carruthers et al, 1976).

Reduced oxygen tension or reduced atmospheric pressure has been suggested as aetiological factors for DVT or PE (Carruthers et al, 1976). It is unlikely that they do, otherwise an increased incidence of DVT in people living at altitudes or in patients with hypoxia would be reported.

On the other hand majority of patients with traveller's thrombosis appear to have other risk factors. At least one risk factor is present in 75-80 per cent of cases (Kesteven, 2002). The most common risk factors are previous DVT, malignancy, hormone treatment or recent surgery. This is consistent with Rosendaal's multi-hit theory in the aetiology of DVT (Rosendaal, 1999).

The association between air travel and DVT was documented by Ferrari et al 1988) in a retrospective case control study. The researchers concluded that a history of recent air travel is found almost four times more frequently in the DVT group and the odds ratio in patients who travel is 3.98.

Literature and publication like Ferrari et al (1988) lend support to the hypothesis that venous thrombosis is associated with air travel but carefully controlled studies are needed to test this association properly. Most of the studies give no details of the number of thromboembolic episodes from which those associated with travel were drawn. The cases described by Symington and Stack (1977) represented only 8 out of 182 cases of PE over three years, suggesting that travel is not a major cause.

In a retrospective study of 254 patients, Eklof et al (1996) identified 44 patients (24 males and 20 females) with a mean age of 63 years, who presented with venous thromboembolic complications. Flight times were 5-17 hours. Seven cabin-related and five patient-related risk factors were identified.

The seven cabin-related risk factors are listed below:

- Low humidity
- Dehydration
- Diuretic effect of alcohol
- Smoking
- Hypoxia
- Immobilisation
- Coach position

The five patient-related risk factors present in varying percentages are tabulated below:

Table 2.14: Patient-related risk factors in traveller's thrombosis

Patient-related factors	Percentage
Previous DVT	34
Malignancy or chronic diseases	25
Hormone Replacement Therapy	16
Recent lower limb injury	11
Recent surgery	9

Data: Ecklof et al, 1996

Although the patient-related risk factors may be potentiated by the cabin-related risk factors, Ecklof et al (1996) concede that further work is required to support this assumption and concluded that modern air transportation is safe.

There is no prospective epidemiological data to support the association between air travel and venous thrombosis, current evidence is weak and traveller's thrombosis is much less common than the impression created by recent publicity (Geroulakos, 2001).

Summary of theoretical framework

The review of the theoretical framework in the epidemiology of DVT has provided an understanding of the nature and magnitude of the problem. A discussion of the risk factors has also highlighted a theoretical model underpinning the clinical assessment of patients at risk of DVT. Such clinical data should be seriously considered in hospitalised patients. The risk factors are of practical value in predicting which patients are most likely to develop DVT. The data can also be used to stratify patients into DVT risk categories. Surgery, trauma, high risk diseases, advancing age, immobility, pregnancy and puerperium, oral contraceptives and other miscellaneous factors have been discussed in terms of their potential causation of DVT. Those risk factors have been examined to point out the commonality of the Virchow's postulates and to justify their inclusion in the development of the Autar DVT risk assessment scale in chapter three.

Chapter Three

Development of the Autar DVT risk assessment scale

Risk generally refers to the probability of some untoward event. It is restricted to describe the likelihood that people who are without a disease, but are exposed to certain factors, will acquire the disease. Risk factors are those characteristics or events that have been shown to increase the probability of becoming diseased. While risk factors are indicators of an increased probability of a specific outcome, they may or may not be directly related to the cause of the health problem (Harkness,1995).

Risk assessment scales are developed to measure an individual's degree of risk. The recognised risk factors are translated into some quantifiable data, which identify the nature and degree of risk. Risk assessment facilitates the application of early and timely intervention that minimises potential complication or at the very best, prevents its occurrence completely.

An ideal risk assessment tool has high sensitivity and specificity. Risk assessment tools have practical application and enable a systematic approach to risk assessment. However, risk assessment tools are no substitute for clinical decision making, based on continued observations and what the patient says, for this is often the best evidence (Castledine, 1997). Clinical and professional judgement supplements the application of a risk assessment tool. Although the Autar DVT risk assessment scale can stand alone, its consistency and predictive accuracy can be further enhanced by the complementary effect of clinical decision making (Capper, 1999). For example, it does not necessary follow that an immobile patient on complete bed rest is unable to perform some ankle flexion and dorsiflexion exercise to promote venous return. In such a case, the nurse assessor must use the DVT scale as designed but also make written observation of the ability of the patient to execute simple instructions and carry out some form of ankle exercise.

Prediction or probabilities can guide clinical decision-making (Altman 1994). Even when the prediction does not come true in an individual patient, it will usually be borne out in many cases (Fletcher, Fletcher and Wagner, 1996).

Patients' DVT risk assessment status does not usually remain static and fluctuates within a continuum of no risk to high risk (Table 3.1).

Table 3.1: DVT risk continuum

Risk categories	Risk percentage
No risk	0
Low risk	< 10
Moderate	11-40
High risk	41>

Data: Salzman & Hirsh, 1982; Hull et al, 1986; Caprini et al, 1988; Anderson & Wheeler, 1995.

DVT prognostic indices

The Autar DVT scale was developed and named after the investigator to differentiate it from the other DVT prognostic indices chronologically listed below:

- Nicolaides and Irving, 1975
- Clayton et al, 1976
- Lowe et al, 1982
- Sue-Ling et al, 1986
- Janssen et al, 1986
- Rocha et al, 1988
- Arcelus et al, 1991
- Caprini et al, 1991
- Bahal & Silverman, 1993

Extensive search and review of medical literature have highlighted the plethora of prognostic indices listed above for the identification of patients at risk. Nicolaides and Irving (1975) devised a method that could determine the risk of DVT in patients before surgery without the need for special haematological tests. Using a multivariate analysis and logistic regression, they identified age, varicose veins, history of DVT and PE, premedication with Omnopon, obesity and severity of operation as discriminating clinical risk factors.

Clayton et al (1976) devised a simple prognostic index for predicting which patients would develop postoperative DVT. It comprised five variables deemed to be of the best predictive power which were:

- Euglobulin (ELT)
- Age (Yrs)
- Presence of Varicose Veins (Var)
- Fibrin Related Antigens (FRA)
- Percentage Overweight (OWT)

The equation of the prognostic index is:
I: Index = -11.3 + 0.009 (ELT) +0.085 (Age) +2.19 (Var), 1 when present and 0 when absent) + 0.22 (FRA) + 0.043 (OWT).

Validated on 124 patients undergoing major gynaecological surgery, the prognostic index identified 95% of those who developed DVT and misallocated 28% of those who did not develop DVT.

Further to confirm the validity of the Clayton predictive index, Rakoczi et al (1978) tested it on patients undergoing abdominal hysterectomy for malignant diseases. A cutoff score of +4 for the equation identified 8 of 9 patients with DVT. The promising reported results generated considerable interest into further validation of the tool. Crandon et al (1980) investigated 62 consecutive patients aged 40 years or older undergoing major gynaecological procedures by either vaginal or abdominal routes. The predictive index identified 9 out of 10 patients correctly and incorrectly identified 7 out of 52 patients.

Lowe et al (1982) modified and applied the Clayton et al index to 144 patients aged 40 and over undergoing gastrointestinal surgery. The five variables were age, weight, presence of varicose veins, sex and cigarette smoking, plus a battery of non routine laboratory variables such as Plasma viscosity, Blood viscosity, Plasminogen, Fibrinogen, Red Cells Deformability.

Using stepwise logistic discriminant analysis, Sue-Ling et al (1986) identified 7 out of 18 clinical and laboratory variables to construct an index for preoperative prediction of patients at high risk of DVT after major elective abdominal surgery. The seven discriminating variables were age, ELT, previous abdominal surgery, varicose veins, antithrombin III concentration,

cigarette smoking and platelet count. Preoperatively, the predictive index correctly identified 91% of patients in whom DVT developed and wrongly allocated to the high risk group 19% of those in whom it did not. The application of successful DVT prophylaxis depends on the ability to identify high-risk patients. To this end, Janssen et al (1987) devised an interactive computer programme to aid physicians' predictive power and not to preclude their clinical judgement. The programme comprised of 21 risk factors drawn from the literature. Each of the factors was assigned a relative risk based on available data.

Rocha et al (1988) also used a stepwise logistic discriminant analysis to identify preoperative DVT predicting factors. The three laboratory variables identified were Fibrinogen Degradation Product (FDP), Plasminogen Activator Inhibitor (PAI) and tissue Plasminogen Activator (t-PA). The laboratory variables were measured in one hundred and eleven patients and achieved 100% sensitivity and 95% specificity.

Laboratory variables are far from being user-friendly and in the interest of reproducibility, robustness and ease, Ruckley (1985) suggests that simple clinical criteria could be used to identify those at risk of DVT. Such clinical features would fit automatically into ward routine and are much more likely to be consistently and successfully applied. In the footsteps of Ruckley (1985), Caprini et al (1991) and Arcelus et al (1991) developed a risk assessment score sheet based on 20 clinical risk factors for assessment of surgical and medical patients respectively (appendix 1). The index was derived from primary and secondary hypercoaguable states that are frequently associated with DVT. No validated data were reported as to the efficacy of the two predictive indices.

Bahal and Silverman (1993) developed a DVT risk assessment sheet based on 13 recognised risk factors. A scoring protocol identified the high risk category. No predictive data were available, although its evaluation audited a high uptake of prophylaxis when a formal risk assessment chart was in place.

Numerous problems have been identified with these prognostic indices. The frequently tested Clayton et al (1976) index was biased towards a

favourable result for their own types of patient from whose data it had been originally derived.

The instruments developed by the various investigators are dissimilar both in terms of contents and the weighting of the risk factors. The equations derived to calculate the cutoff scores are forbiddingly complex and the haematological variables have tended to be specific to their home laboratory (Gallus, 1989). Lack of standardisation in the laboratory procedure for estimating ELT inevitably accounts for inconsistency in the results. For example, the higher cutoff point used by Rokoczi (1978) resulted from longer ELT. Although Fibrinogen estimation in the detection of DVT is cheap and widely available, there is an urgency to standardise methodology or establish an international standard. Palareti et al (1991) found that calibrated procedure proposed by the manufacturer of a standard analysed kit was not always precise. Poller and Taberner (1982) also reported an international variation in the mean Warfarin dose due to the different types of thromboplastin tissue used in blood clotting tests.

Lowe (1993) concedes that some of the haematological variables are more predictive of arterial thrombotic events than venous thromboembolism. Arterial thrombosis is associated with atherosclerosis whereas venous endothelial damage is implicated in DVT (Rosendaal, 1997).

Some of the clinical variables identified as discriminating risk factors are inexplicable and may have occurred by chance. Lowe's index (1982) identification of smoking as a risk factor shows no positive association with DVT (Prescott et al, 1978, Meade, 1983). Nicolaides and Irving (1975) identify omnopon premedication as an independent risk discriminant, without any explanatory biological foundation.

The Janssen et al (1987) interactive computer programme based on 21 relative risk factors was derived from out of date data. Some risk factors were identified but not assigned any relative risk value. For example, it is generally acknowledged that varicose veins are a risk factor, albeit direct or indirect in nature. However, the computer interactive programme did not assign any relative risk percentage to varicose veins.

Because many of the prognostic indices were developed for a specific clinical area, they varied in contents, cutoff scores and practical application and therefore lacked transferability. The Clayton et al index used in gynaecological surgery is not readily applicable to other general surgery. Similarly, although the Rocha et al index (1988) achieved high sensitivity, arguably, the predictive index for an already high risk group such as total hip replacement, does not permit generalisation of findings. Additionally, as illustrated in table 3.2, five investigators have used different cutoff levels for the same predictive index, with varying outcomes.

Table 3.2: Cutoff levels for Clayton et al predictive index

Investigator	Cutoff level Where I = Index	Sensitivity %	Specificity %
Clayton et al,1976	I= -2.5	95	72
Crandon et al,1980	I= -2.0	90	87
Rokoczi et al, 1978	I= -2.5	100	20
Lowe et al, 1980	I= 1.75	90	52
Melbring & Dalgren, 1983	I= 1.75	76	27
Sue-Ling et al, 1986	I= -1.5	91	69

Adapted from: Gallus, 1989

Lowe et al (1982) developed a simple index based on age and body weight and claim that it was a useful basis for selecting DVT prophylaxis in gastrointestinal surgery. When Melbring and Dahlgren (1983) looked for DVT in similar patients, they found that the Lowe index had low sensitivity and specificity and concluded that it did not work (table 3.2). An instrument that is transferable to a number of settings is likely to yield more information not only about its reliability and validity, but also about the practicalities, appropriateness, acceptability and suitability of the instrument (Gibbon, 1998).

Although a few predictive indices have shifted their focus from laboratory variables to essentially clinical risk factors to eliminate delay in decision making, they are not readily understood in terms of why some individuals with risk factors develop DVT relative to those who do not.

The majority of the assessment strategies available to predict patients at risk of DVT continue to be laboratory-dependent, expensive, invasive in nature

and remain the prerogative of medical staff. Nurses are well placed to manage proactively the DVT risk assessment and its primary prevention. Two audit studies have demonstrated that when nurses are actively involved in risk assessment of patients, the uptake of venous prophylaxis is increased by 50 percent (Bahal and Silverman, 1993, Byrne et al 1996). A risk assessment tool that is easy to understand and apply, user friendly and inexpensive is more likely to be accepted by everyone. It can make a major contribution to further harmonisation between the clinical roles of nurses and doctors to enhance the notion of collaborative care. One such assessment tool was developed to assess the level of consciousness by Teasdale and Jennett (1974). The popular and well-established Glasgow Coma Scale devised to monitor altered level of consciousness, is effectively applied by nurses and doctors alike. Its psychometric properties are easily understood and the coma scale is now universally accepted.

The need for a generally applicable scale for assessing patients at risk of DVT is clinically justified on the following arguments:

- Clinical diagnosis of DVT is notoriously unreliable even to the most watchful observer and there is general agreement on the need to assess patients to guide decision making (Prandoni and Mannucci, 1994; THRiFT, 1998). Barnes (1982) found approximately 50% inaccuracy when using the Homans' sign to make a diagnosis of DVT. In Homans'sign, pain is elicited on dorsiflexion. Two thirds of venous thrombi are silent (Salzman, 1986) and only 3.5 % of patients develop clinical symptoms (Caprini and Natonson, 1989).
- Assessment of patients into DVT risk categories enables the implementation of the most appropriate venous thromboprophylaxis strategies, as recommended by consensus groups.
- A formalised risk assessment protocol increases the uptake of venous thromboprophylaxis (Bahal and Silverman, 1993, Byrne et al, 1996).
- There are marked inconsistencies in the existing system for assessing patients at risk of DVT. A diagnosis of potential problem of DVT provides a very crude index of risk assessment. A systematic and objective assessment of risk factors should be performed on each patient to facilitate a management plan (Paiement and Mendelsohn, 1996). Unless

the degree of risk is precisely identified, venous thromboprophylaxis is likely to be ineffective.

- There is a lack of an organised strategy for DVT risk assessment, resulting in ambiguities and misunderstandings during communication and decision-making (Vanek, 1991).

Objectives of the Autar DVT scale

The Autar DVT scale (1994,1997) as illustrated in figures 1 and 3 was developed to:

- provide objective evidence of risk status
- provide a DVT risk calculator that is simple to be used equally reproducibly by nurses, medical staff and other health care professionals
- provide a comprehensive assessment of patients at risk of DVT
- calculate the degree of risk in an individual, in the context of multifactoral aetiology, across diverse clinical areas.
- provide consistency in the clinical assessment of practice
- provide evidence to support decision making and practice
- enable the application of preventative measures, commensurate with the category of risk
- allow the targeting of limited resources
- provide quantifiable data for auditing purposes and quality assurance
- develop a body of clinical nursing knowledge, embedded in practice.
- provide a teaching tool for the understanding of the pathophysiology of DVT.
- empower nurses within the scope of professional practice (UKCC 1992, Autar,1996 b) and the Higher Level of Practice (HLP) framework proposed by the UKCC (2001). With the inception of Advanced Nursing Practitioners, nurses are now exploring new territories and developing new roles in the interest of enhancing patient care. HLP has already contributed to the appointment of Nurse Consultant and is already being used to structure job description, inform life long learning and support risk management.

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Figure three Autar DVT Risk Assessment Scale (Revised 1997)

ADDRESSOGRAPH

Name:

Unit No:

Ward:

AGE SPECIFIC GROUP

Age Group	Score
10-30	0
31-40	1
41-50	2
51-60	3
61+	4

BUILD BODY MASS INDEX (BMI)

Build	BMI	Score
Underweight	16-19	0
Average	20-25	1
Overweight	26-30	2
Obese	31-40	3
Very Obese	41+	4

$BMI = wt(kg)/ht(m)^2$

MOBILITY

Risks	Score
Ambulant	0
Limited (uses aids self)	1
Very limited (needs help)	2
Chair bound	3
Complete bed rest	4

SPECIAL RISK CATEGORY

Oral contraceptive:	Score
20-35 yrs	1
35+ yrs	2
Pregnancy/Puerperium	3

ASSESSMENT PROTOCOL

Score ≤6	No risk
Score 7-10	Low Risk (<10%)
Score 11-14	Moderate Risk (11-40%)
Score 15≥	High Risk (>41%)

THROMBOPROPHYLAXIS

Please tick the appropriate prescribed DVT prophylaxis

- **Leg Elevation** ☐
- **Graduated Elastic Compression Stockings (GECS)** ☐
- **Pneumatic Compression (PC)** ☐
  - Intermittent or ☐
  - Continuous ☐
- **Heparin** ☐
  - Heparin Sodium (standard/unfractionated)
  - Calciparine (heparin calcium)
  - Minihep (heparin sodium)
  - Minihep Calcium (heparin calcium)
  - Monoparin (heparin sodium)
  - Monoparin Calcium (heparin calcium)
  - Multiparin (heparin sodium)
  - Pump-Hep (heparin sodium)
  - Unihep (heparin sodium)
  - Uniparin (heparin sodium)
  - Uniparin Calcium (heparin calcium)
- **Low Molecular Weight Heparin** ☐
  - Fragmin (dalteparin sodium)
  - Clexane (enoxaparin Heparin)
  - Innohep (tinzaparin)
  - Logiparin (tinzaparin)
- **Warfarin** ☐
- **Others, please specify:** ☐
- **No Prophylaxis** ☐

TRAUMA RISK CATEGORY

Score item(s) ONLY pre-operatively	Score
Head	1
Chest	1
Spinal	2
Pelvic	3
Lower Limb	4

SURGICAL INTERVENTIONS

Score only ONE appropriate item	Score
Minor Surgery (<30 mins)	1
Major Surgery	2
Emergency Major Surgery	3
Thoracic	3
Abdominal	3
Urological	3
Neurosurgical	3
Orthopaedic	4

HIGH RISK DISEASES

	Score
Ulcerative Colitis	1
Anaemia: Sickle Cell	2
Polycythaemia	2
Haemolytic	2
Chronic Heart Disease	3
Myocardial Infarction	4
Malignancy	5
Varicose Veins	6
Previous DVT or CVA	7

SCORING (within 24 hours of admission)

Identify appropriate items, add and record score below

Assessor	Date	Score

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The Autar DVT scale is based on Virchow's triad in the genesis of DVT. In the selection of the risk factors to prospectively identify patients prone to DVT, the dependent and explanatory variables are considered. The dependent variable is DVT and the explanatory variables are the risk factors whose presence or absence is associated with occurrence of DVT (Sigel et al, 1974).

In the consideration of the risk factors for DVT the following criteria are taken into account:

- The natural history of DVT
- Initial selection of risk factors
- Relative risk.

For the purpose of this study, the natural history of DVT has been divided into three stages, namely:

1. **Susceptibility**: This relates to the presence of relatively permanent factors, which increase the likelihood of DVT in an individual. Factors as age, sex and persisting or permanent disease such as chronic heart failure, CVA and varicose veins are example of these.

2. **Predisposing factors**: These are factors which are often reversible but which may initiate and enhance the development of DVT. When these effects are diminished, there is a reduction in the likelihood of DVT. Trauma, immobility, obesity, surgical interventions and high-risk diseases such as ulcerative colitis, acute myocardial infarction and malignancy are some contributory factors.

3. **The clinical condition**: This refers to patients with a previous history of DVT who may be at high risk for the development of further DVT episodes. Samama et al (1993) claim that patients with DVT are eight times likely to have recurrence of the condition.

Initial selection of risk factors: This relates to the most independent and discriminating predictors of DVT. In the selection of the best predicting risk factors, the investigator was guided by the review in the literature and by generally accepted clinical tenets. Additional information was also available from the pilot study previously undertaken (Autar, 1994).

Relative risk: The effect of a risk factor can be measured by means of an odds ratio or relative risk (Fleiss, 1973). The relative risk of a factor depends on the incidence of DVT in patients with and without that variable. Relative risk is independent of the actual frequency of DVT in different population. For example, a patient who is immobile is seven times more likely to develop DVT than one who is fully ambulant (Samama et al, 1993).

The Autar DVT scale is conceptualised on the triad of aetiological factors postulated by Virchow, which are fully addressed in chapter two.

The DVT scale is composed of seven distinct subscales of risk factors drawn from the extensive literature review from nationally and internationally recognised authority (NIH, 1986; SIGN, 1985; International Consensus Statement, 1997; THRiFT 11, 1998).
The seven parameters of the subscales are illustrated in table 3.3.

Table 3.3: DVT subscales.

DVT subscales	Item rating
Age Specific Group	0 – 4
Build / Body Mass Index (BMI)	0 – 4
Mobility	0 – 4
Special Risk Category	1 – 4
Trauma Risk Category	1 – 4
Surgical Intervention Category	1 – 4
High Risk Diseases	1 – 7

Data: Autar, 1996 a; Autar, 1997

The descriptors or options in each subscale are assigned a risk score in the light of their relative risk and potential for initiating DVT. A value of 0 is assigned to no risk and a score of 1 to the lowest risk factor. Other risk factors are calculated relative to this value based on previous studies and the incidence of DVT. Independent and high risk factors in the genesis of DVT are weighted at a score of 4 or more.
It is the purpose of this section to draw on the most relevant reviewed theoretical framework and biological basis of DVT to highlight the rationale underpinning the selection of the well-recognised risk factors and their clinical justification as covariates of the Autar DVT scale.

Age specific group

Kakkar et al (1970) and Nicolaides & Irving (1975) reported a strong correlation between advancing age and the risk of DVT. In their prognostic indices developed to identify patients prone to DVT, Clayton et al (1976); Lowe et al (1980) and Sue-Ling et al (1986) have convincingly demonstrated that increasing age is not just a significant risk factor but also has a powerful predictive power.

The age specific group subscale comprised of five distinct age groups (Table 3.4).

Table 3.4: Age specific group

Age group	Item rating
10-30	0
31-40	1
41-50	2
51-60	3
61+	4

Young age appears to have a protective effect against DVT (Rohrer et al, 1996). The descriptor 10-30 age group of the subscale is assigned a score of 0 to indicate that patients in this group are not at risk. Incidence of DVT is very rare in children (Jones & McIntyre, 1975) and an incidence of 7 per 100,000 children per year has been estimated (Hirsh & Hoake 1996; Rosendaal, 1997). Young patients who develop DVT invariably have strong risk factors (Coon 1976; Berstein et al,1986; David & Rivard,1991).

Incidence of DVT rises after the age of 30 (Coon, 1976) and Janssen et al (1987) computed a relative risk of 1 for patients over 35 years old. Equally, a weighted score of 1 is allocated to the 31-40 age group of the subscale.

The risk of DVT rises sharply after the age of 40 (Caprini et al, 1991; THRiFT, 1992). Kakkar et al (1970) reported a DVT incidence of 24 % in patients between the age 41-50 and 47% for those in the 60-80 years old. Samama et al (1993) claim 50% of DVT occurs in patients in their 60-80's. As DVT increases exponentially with advancing age (OPCS 1990;

Rosendaal, 1997) descriptors 51-60 age group and 61+ are assigned the relative weighted score of 3 and 4 respectively.

Build / Body Mass Index (BMI)

In this subscale, patients are broadly classified into one of the five groups of builds, according to their body mass index (Table 3.5).

Table 3.5: Build / Body Mass Index (BMI)

Build	Item rating
Underweight	0
Average (normal / acceptable)	1
Overweight	2
Obese	3
Very Obese	4

Build is a subjective term and is often ill-defined in clinical studies (Anderson et al, 1995). BMI offers a simple and objective assessment of patients' builds derived from the formula:

BMI=Weight (kg) / Height (m^2)

Another major advantage of the BMI is that the standards are applicable to both male and female patients. A BMI build conversation chart is landscaped on the reverse of the DVT scale data collection tool, to facilitate quick and ready reckoning of the build (figure four: p 108).

The incidence of DVT in overweight patients is 47.9% compared to 21.7% in non-obese patients as illustrated in table 3.6.

Table 3.6: Incidence of DVT by build

Build	DVT incidence %
Underweight	21.7
Average	27.2
Overweight	47.9

Data: Kakkar et al, 1970.

There is an association between obesity and the risk of DVT but its significance disappears when other risk factors present in patients are considered (Nicolaides & Irving, 1975). Coon (1976) also reports that obesity increases the risk of DVT and PE between 1 to 5 times that of the general patient population. Noting the finding of Coon (1976) Janssen et al (1987) assigned a 1.5 times greater risk of DVT for obese than non obese patients, in their interactive computer risk assessment programme.

Although the effect of obesity on the risk of DVT is inconclusive and questionable, Anderson et al (1995) concede that it is only an additive marker, not necessitating prophylaxis in itself.

From the physiological perspective, obesity is associated with diminished venous return (Christopoulos et al, 1980; Mammen 1992). Some impairment in their fibrinolytic system has also been reported (Vanek, 1991; Poller, 1993).

In the light of available data, in this subscale, the underweight patients have a rating of 0 and a score of 1 assigned to the average build. The obese and grossly obese patients who are three times at risk of DVT (Samama et al, 1993) are allocated a score of 3 and 4 respectively. The assignment of a score range of 0 for the underweight build and 4 for the very obese is also harmonious with the grading system of build and Body Mass index (Table 3.7)

Table 3.7: Grading system for Build and Body Mass Index

Grading system for build and BMI

Grade	Build	Body Mass Index (BMI)
0	Underweight	16-19
1	Average/ desirable	20-25
2	Overweight	26-30
3	Obese	31-40
4	Very obese	41+

Data: Garrow & Webster, 1985

Mobility

There is a striking relationship between immobility and the incidence of DVT (Gibbs, 1957; Warlow, 1978; Kierkegaard et al, 1987). In the mobility subscale, patients were identified into one of the five functional levels of classification (Table 3.8).

Table 3.8: The five levels of functional mobility.

Mobility	Item rating
Ambulant	0
Limited (uses aids, self)	1
Very limited (needs help)	2
Chair bound	3
Complete bed rest	4

Immobility causes impairment of the venous pump and stasis which is one of the three elements in Virchow's triad predisposing to DVT. It is generally recognised that early mobilisation minimises the risk of DVT (Rose,1979; Kierkegaard et al, 1987). The old and trusted prophylactic approach to DVT is early ambulation. During weight bearing, a venous plexus in the sole is rapidly emptied into the deep vein of the leg. This flow produced by mobilisation, flushes out the valve pockets in which thrombi are thought to originate (Gardner & Fox, 1983).

The ambulant patients are judged not to be at risk and are assigned a zero score. Patients who have limited mobility and use aids (self) are given a score of 1 and for those who have very limited mobility and require the assistance of another person, a relative risk score of 2 is deemed appropriate.

Poor sitting position causes venous stasis (Singer, 1983; Schaubs et al, 1984). The chair bound patients were assigned a score of 3 as "prolonged sitting position occasions a degree of dependency stasis", resulting in the formation of DVT (Homans, 1954). Sitting in a chair or on the bed with the legs dangling dependently serves to worsen stasis and increases the risk of DVT (Rose, 1979).

Kierkegaard et al (1987) reported a DVT incidence of 13 percent in non-surgical bedridden patients. In immobilised multiple trauma patients, a DVT incidence of 69 percent has been recorded (Kudsk et al, 1989). The frequency of DVT following complete bed rest is mainly due to the scarce utilisation of the calf muscle pump with venous stagnation in the lower limbs (Gibbs, 1957). Confinement to bed is synonymous with muscle inactivity (Kaisary, 1980) and the completely bed ridden patients are judged to at high risk of DVT and fittingly assigned a score of 4.

Special risk category

Hypercoagulability is the most pronounced element of Virchow's triad present in this special risk category. This subscale comprises aetiological risk factors such as oral contraceptives and pregnancy and puerperium (Table 3.9).

Table 3.9: Special risk category

Special risk factor	Item rating
Oral Contraceptives: 20-35 years old	1
Oral Contraceptives: 35+ years old	2
Pregnancy and Puerperium	3

In patients taking oral contraceptives, Coon (1977) documented an increased probability of clotting problems due to:

- Increased vascular endothelium permeability.
- Reduced venous tone.
- Reduced blood flow velocity
- Increase in clotting factor levels.

The relative risk of oral contraceptives depends on the dose of oestrogen (Stadel, 1981). The risk of DVT is twice as high with "third generation" oral contraceptives containing desogestrel and gestodene (WHO, 1995).

Sartwell et al (1969) attributed a risk factor of 4.4 times as the average value for oral contraceptives. However, a 50 microgram dose of oestrogen is arbitrarily assigned a risk of 1, 100 microgram dose as causing 3.2 times increased risk and 150 microgram as resulting in 5.9 times increased risk

(Vessey & Inman, 1973). On the other hand, lowering the dose of oestrogen (Anderson et al, 1995) has markedly reduced the risk of DVT associated with oral contraceptives.
Among women taking oral contraceptives, Vessey and Doll (1970) reported 2.5 times increased incidence of DVT in those aged 35-44 group over women in the 20-34 group. Noting this finding, the women in 20-34 age group are assigned a score of 1 and a relative score of 2 used for the over 35 years old.

The increased risk of DVT during pregnancy and puerperium period is approximately 5.5 times that of nonpregnant women who are taking no oral contraceptive (Coon, 1977). Natural haematological changes occur during pregnancy. Fibrinogen levels in the blood in late pregnancy are at least double the levels found in the nonpregnant state (Letsky, 1989). As illustrated in table 3.10, pregnancy is a more risky option than taking the combined oral contraceptives (Allotey and Louca, 1997) and this risk factor is assigned a relative risk score of 3.

Table 3.10: Annual DVT Incidence per 100,000 women

Risk factor	DVT cases
Non pregnant and no oral contraceptive	5
Combined oral contraceptive	15
High risk combined oral contraceptive (Third generation contraceptives)	30
Pregnancy and Puerperium	60

Data: Family Planning and Reproductive Health Care,1995

Trauma risk category

Varied reports on multiple traumas and the diverse nature of the injuries have made it very hard to interpret the data. Shackford et al (1990) claim that the incidence of DVT in trauma patients has been artificially over estimated. This is largely due to the inclusion of an elderly population of patients with hip fractures who are already a high risk group. However, in a

prospective study of venous thromboembolism after major trauma, Geerts et al (1994) found DVT in 47% of the patients.
The Trauma Risk Category subscale is represented by five musculo-skeletal injuries (Table 3.11).

Table 3.11: Trauma risk category subscale

Trauma risk factor	Item rating
Head injury	1
Chest injury	1
Spinal injury	2
Pelvic injury	3
Lower limb injury	4

Coon (1976) claims that all patients with accidental trauma, irrespective of sites, are at risk of DVT. Although head and chest injuries are associated with DVT development, their actual percentage is not significantly higher than that expected in the general patient population. Head and chest injuries carry 2.5% risk each and it increases to 14% for spinal injury. An average incidence of DVT of 38% is associated with spinal injury (Geert et al, 1994), although varied reports in literature have documented DVT, ranging from10 to100% depending on the method of evaluation (Merli et al, 1988). The estimated risk for pelvic injury is 22% (Coon, 1976) and DVT was found in 56% of trauma patients (Geerts et al, 1994). Lower limb trauma patients are a high risk group and DVT incidence ranges between 77-80% for tibial and femoral fractures (Geerts et al, 1994, Turpie, 1997). Risk of DVT is calculated at 1.4 times for spinal fractures, 2.7 times for pelvic fractures and approximately 5 times for tibial and femoral fractures (Janssen et al, 1987).

Based on the available data on trauma, the muscular skeletal trauma items are scaled according to their ascending percentage of risk and potential for causing DVT.

Head and chest injury is each apportioned a score of 1. Spinal and pelvic injuries are rated a risk score of 2 and 3 respectively. All three elements of Virchow's triad are present in lower limb injury and this item is affiliated with a score of 4. In multiple trauma patients, more than one risk factor may be present, in which case, the risk scores are summated.

The trauma risk category subscale is applicable to patients in both the surgical and orthopaedic specialities. Head and chest injury patients are more likely to be admitted to the surgical directorate and spinal, pelvic and lower limb injuries to the orthopaedic and trauma unit.

It is recommended that this subscale is recorded only preoperatively and is ignored as and when the patient has the appropriate surgical procedure in the surgical intervention category of the DVT scale. The surgery indicated for the trauma by its very therapeutic and corrective nature cancels the trauma risk category. For example, a patient with a hip fracture would initially be assigned a score of 4 from the Trauma subscale but this would then be substituted by a new score of 4 for the orthopaedic surgery in the surgical intervention subscale. Concurrent scoring of both the trauma risk category and surgical intervention subscales inflates the aggregate score, over estimates the risk, misclassifies the patient and consequently predicts a false positive (Autar, 1994).

Surgical intervention category

It is well recognised that DVT is a common postoperative complication (Lindblad, 1991) and surgery is the leading risk factor (Moser, 1989). All the three elements of Virchow's triad are interactive: venous stasis, hypercoagulability and vessel trauma.

The surgical intervention category subscale comprises eight surgical options, ranging from minor surgery, posing low risk of DVT to orthopaedic surgery, notorious for its very high incidence of DVT (Table 3.12).

Table 3.12: Surgical intervention category subscale

Type of surgery	Item rating
Minor surgery (< 30 mins)	1
Major surgery	2
Emergency major surgery	3
Thoracic surgery	3
Gynaecological surgery	3
Abdominal surgery	3
Urological surgery	3
Neurosurgical	3
Orthopaedic surgery	4

Only one appropriate surgical option is applicable to patients. The options cover a wide range of surgical specialities, signifying the universal applicability of the DVT scale.

There is a positive correlation between the duration of surgery and the incidence of DVT (Borrow and Goldson, 1981; Moser, 1989). The performance of a surgical procedure on a patient requiring more than 30 minutes of general anaesthesia is a critical risk factor. Gouke (1989) defines major surgery as a procedure requiring general anaesthesia for more than 30 minutes. This definition has serious implication for even a relatively simple procedure requiring 30 minutes of anaesthesia and places the patient at risk. Flanc et al (1969) first reported an incidence of 44 percent of DVT for major surgery, in comparison to a low 15 percent for minor surgery. Elderly patients in their 60-80's undergoing minor surgery have a DVT incidence of 31.6 percent compared to 51 percent for major surgery matched for the same age group (Kakkar et al, 1970).

Appropriately, minor surgery is weighted at a low risk score of 1 and 2 assigned to major surgery. Incidence of DVT ranges from 0.2-2.2 percent for elective major surgery compared to 2.7 percent for emergency major surgery and the latter is given a risk score of 3.
The overall frequency of thromboembolic complications after thoracic, gynaecological, abdominal, urological and neurosurgery is relatively of the same magnitude (International Consensus Statement, 2001). It is for this reason that they are all assigned a risk score of 3(Table 3.12).

Some surgical procedures are associated with comparatively higher incidence of DVT than others. The risk following below waist orthopaedic surgery is greater due to manipulation of the leg causing distortion and occlusion of the femoral vein (Stamatakis et al, 1977). One in two patients undergoing total hip replacement develops DVT (Parker-Williams and Vickers, 1989). Table 3.13 illustrates the incidence of DVT with different types of surgery.

Table 3.13: Association between types of surgery and incidence of DVT

Types of surgery	DVT incidence (%)
Orthopaedics: Knee arthroplasty Hip fracture repair Hip arthroplasty	 75-84 60-70 30-65
AbdominoThoracic	26-33
General abdominal	3-51
Gynaecological	7-45
Urological: Transvesical prostatectomy	21-51
Neurosurgery	29-43

Data: Becker, 1986; Das, 1994 and Zahn et al, 1999

Proportional to the average incidence of DVT associated with the types of surgery, thoracic, abdominal, gynaecological, urological and neurosurgery are all assigned a rating risk score of 3. All below waist orthopaedic procedures carry a risk score of 4.

High risk diseases category

In this subscale, ten high risk diseases are represented (Table 3.14).

Table 3.14: High risk diseases

High risk diseases	Item rating
Ulcerative colitis	1
Anaemia: Sickle cells	2
Polycythaemia	2
Haemolytic	2
Chronic heart disease	3
Myocardial infarction	4
Malignancy	5
Varicose veins	6
CVA	7
Previous DVT	7

One or more risk factors may be present in a patient. This subscale is not primarily confined to the medical directorates as such pre-existing illnesses are commonly encountered in many other clinical areas. The evidence that

significantly more than half of the postoperative DVT is present prior to surgery emphasises the importance of the pre-existing diseases as a cause of DVT (Wright et al, 1951, Sevitt and Gallagher, 1969). One of the three elements of Virchow's triad is implicated in the high high risk diseases. Ulcerative colitis, the anaemias and malignancy are characterised by increased in procoagulants. The cardiovascular disorders such as chronic heart failure and myocardial infarction cause venous stasis and intimal vessel wall changes and damage to the valves are evident in varicose veins and previous DVT. The association between the incidence of DVT and the high risk diseases is illustrated in the following studies: (Table 3.15).

Table 3.15: The association between high risk diseases and Incidence of DVT

Study	Diseases	DVT %
Edward and Truelove,1964 TrueLove,1984 Wyshock et al,1988	Ulcerative colitis	1.3-6.4
Shafer,1985	Sickle cell anaemia Polycythaemia vera Haemolytic anaemia	Insufficient and unclassified data
Coon,1977	Heart failure	10-20
Coon,1977, Carter et al, 1987	Myocardial infarction	20-40
Kakkar et al,1970 Browse et al,1977	Malignancy	40-56
Warlow et al,1978	CVA	46-53
Nicolaides & Irving,1975 Schaub et al, 1975 Clayton et al,1976 Sue-Ling et al,1986	Varicose veins	48-56
Nicolaides & Irving,1975 Samama et al,1993	Previous DVT	68

Inflammatory Bowel Disease (IBD) such as ulcerative colitis predisposes to thrombosis. Physiological changes such as an increase in clotting factor VIII and fibrinogen occur in response to haemorrhagic tendency. Edwards and Truelove (1964) first reported the association between DVT and ulcerative

colitis. A risk score of 1 represents the low reported incidence of 1.3-6.4 for ulcerative colitis.

DVT is often the complication of some haematological conditions such as sickle cell anaemia, haemolytic anaemia and polycythaemia vera. Patients with such disorders exhibit haemostatic complications ranging from bleeding to thrombus development (Shafer, 1985). Such patients are therefore cautiously assigned a risk score of 2 due to blood hypercoagulability but there is insufficient data available on their relative risk and potential for causing DVT.

Nicolaides and Irving (1975) reported a 3.8 times greater risk for patients with heart diseases such as atrial fibrillation and congestive cardiac failure. Coon (1977) and Carter et al (1987) have documented a DVT incidence of 10-20 % for chronic heart diseases and 20-40 % for acute myocardial infarction. Chronic heart failure and myocardial Infarction are assigned a risk score of 3 and 4 respectively.

The frequency of DVT goes up two to threefold in patients undergoing surgery for malignancy. The association between DVT and malignancy has some biological basis. Procoagulants have been isolated and fibrinolytic activity reduced, thus predisposing to DVT. Kakkar et al (1970) and Browse et al (1977) reported a DVT incidence ranging from 40-56 percent for malignancy, which is scaled up to a risk score of 5.

Kakkar et al (1970); Nicolaides and Irving (1975) and Coon (1977) have all reported that varicose veins are associated with a twofold increased risk of DVT. Varicose veins are associated with impaired venous return, predisposing to stasis and thrombi formation. Schaub et al (1975) reported a DVT incidence of 56% and that half of the patients who had previous history of varicose veins developed postoperative DVT. In their predictive indices, both Clayton et al (1976) and Sue-Ling et al (1986) found varicose veins to be a good discriminating risk factor. Based on available evidence varicose veins are regarded as a high ranking risk factor by THRiFT (1992) and the European Consensus Statement (1991). A high risk score of 6 is assigned to varicose veins.

DVT of the lower extremities is reported to occur frequently in patients who have had a stroke (Warlow et al, 1978). Within days after a stroke, 46-53 patients develop DVT.

Sioson et al (1981) found that 33% of stroke patients admitted to a rehabilitation hospital develop DVT. DVT occurs in 60-70% of patients with dense hemiplegia (Turpie et al, 1987). A high risk score of 7 is recorded for the Stroke patients, proportional to the high DVT incidence reported. Incidence of DVT is as high as 68% for patients with a previous DVT (Kakkar et al, 1970). Such patients are eight times more likely to have recurrence of their condition (Samama et al, 1993). Previous DVT is the single most powerful independent risk factor (Caprini et al, 1991) and a risk score of 7 marks its very high risk of recurrence.

Other risk factors

Sex, smoking, diabetes and A blood group type have been cited in the genesis of DVT. Women have a higher incidence of DVT than men (Coon, 1976). But when other factors such as oral contraceptives and pregnancy are taken into account, sex difference disappears (OHE, 1996). In the DVT Worcester study (Anderson et al, 1991) reported a higher DVT occurrence in the female patients. But because incidence of DVT increases exponentially with age and women as a group live longer than men, there are more episodes of DVT among women. Epidemiological data analysed indicate that the incidence of DVT is equal for both sexes (Nordstrom et al, 1992).

Smoking has been blamed for many ills and its implication in the causation of DVT is no exception. Analysis of cigarette smoking as a clinical variable for DVT demonstrates a negative association (Clayton et al, 1976, Lowe et al, 1982). Astonishingly, smoking has a protective effect against DVT (Handley and Tether, 1974; Emerson and Marks, 1977; Prescott et al, 1978; Meade 1983; Nordstrom et al, 1992).

Diabetes has been cited as a risk factor but there is little evidence to substantiate such an assumption. Diabetes affects predominantly the arterial vessels, especially those supplying the myocardium and the microvessels of the retina (Banga and Sixma, 1986).

Patients with O blood group type have been reported to have half the incidence of DVT compared to those in group A type (Jick et al, 1969 and Mourante et al, 1971). However, due to the pattern of ABO blood group distribution, the sample in those studies was too small to draw a firm statistical conclusion. Nordstrom et al (1992) found no significant differences in the distribution for blood groups A, B, AB and O types.

Recently, there has been wide publicity surrounding traveller's thrombosis. Economy class syndrome refers to a thromboembolic disease occurring generally after a prolonged air flight. There are plausible physiological reasons why sitting still for a long period might predispose to DVT (Milne, 1992). Ferrari et al (1991) found a history of travel came up almost four times more frequently in their venous thromboembolic disease group. However, most of the reports are anecdotal and the evidence only circumstantial (Geroulakos, 2000). Kesteven (2000) claims that when airline passengers develop DVT or PE, there are other risk factors present. Tardy et al (1993) reported 21 cases of DVT. All but two had risk factors such as obesity (1case) varicose veins (11cases), cancer (1 case) heart disease (10 cases). Current data are extremely limited and no prospective controlled studies have been undertaken.

The evidence associated with sex, smoking, diabetes and A Blood group type and Traveller's thrombosis is inconclusive. The reports are anecdotal and most of the data have not compared the frequency of a risk factor in patients with DVT with its frequency in the total population from which the cases were derived. These items are therefore not considered in the development of the Autar DVT scale.

The guiding principle of the Autar DVT scale is that risk factors are additive and effects, cumulative. The risk factors may combine synergistically to increase the risk of DVT. This principle is consistent with the "multi- hit" theory proposed by Rosendaal (1997) that the several factors combine to provoke an acute episode of DVT. For example, patients who are over the age of 40 years, undergoing a surgical procedure lasting for more than half an hour with coexisting congestive heart failure are high risk group (Gallus et al, 1994). An association is not necessarily a causation of DVT. Major or

independent risk factors have a strong causal consequence, while minor or associated additive risk factors acting singly are of little or no significance. By the same token, the presence of even a strong factor does not mean that the individual is very likely to get the disease (Fletcher, Fletcher and Wagner, 1996). Some investigators have used logistic regression modelling to assign weights to individual risk factors, suggesting that some risk factors are more important (Sue-ling et al, 1986; Clarke-Pearson et al, 1987). For example, a previous DVT is the most important risk factor for DVT. It has an odds ratio of 7.9 for its recurrence and therefore prophylaxis is strongly recommended. On the other hand, women taking oral contraceptive have an additive risk factor in oestrogen. But the use of oestrogen alone is unlikely to present sufficient risk to justify the provision of prophylaxis.

On the other hand, Kakkar et al (1970) and Salzman and Hirsh (1987) have suggested that the incidence of DVT increase in proportion of the number of risk factors present.

The operation of risk factors can usually be understood in terms of one or more elements of Virchow's triad, and when there is no risk factor present, it is very rare although not unknown for patients to develop DVT. In a review of 1231 patients treated for DVT, 96% of the patients had one or more risk factors (Anderson and Wheeler, 1995). The proportion of patients with clinically suspected DVT in whom the diagnosis was confirmed by objective testing increases with the number of risk factors (Table 3.16).

Table 3.16: Association between risk factors and DVT

No of risk factors	DVT%
0	11
1	24
2	36
3	50
>4	100

Data: Anderson and Wheeler,1995

Summation of the cumulative effect of all the risk factors provides a discriminatory and prognostic index to DVT. One high risk factor may be present in low risk category, escalating to 2-4 factors for the moderate risk

group. In the high risk group, more than four factors are present (Caprini et al 1991; Arcelus et al, 1991). 73% of patients who develop DVT have at least two risk factors (Borrow and Goldson, 1981).

To quantify the individual patient's degree of risk, all the risk factors are added up and the patient is classified into one of the four categories according to the total score(table 3.17).

Table3.17: DVT risk assessment protocol

Risk score	Risk category
≤6	No risk
7-10	Low risk
11-14	Moderate risk
≥15	High risk

Most values or tests are not sharply categorised in exact values but exist on a range or continuous spectrum (Topf, 1986). The score range should therefore not replace the professional judgement but it should be another tool to enable nurses to achieve consistency and objectivity in the assessment in practice. However, as professional judgement lacks a gold standard (Brennan & Hayes, 1992) it must be based on factual information, best external evidence and not conjecture or guesswork (Castledine,1997). The best external evidence is usually derived from clinically relevant research based on biosciences, whereas clinical judgement is the individual experience acquired through clinical experience and clinical practice (Sackett et al, 1996). Integrating individual clinical expertise with the compelling evidence of the DVT scale can serve to enhance the predictive accuracy of the risk assessment calculator.

The incidence of DVT and PE associated with each of the risk categories is illustrated in table 3.18

Table 3.18: DVT and PE incidence associated with each risk category

Risk category	DVT incidence %	PE incidence %
Low risk	< 10	0.01
Moderate risk	10-40	0.1-0.7
High risk	41>	1-5

Modified from Salzman and Hirsh, 1982

Low risk patients are those under the age 40 without additional risk factor, who have an uncomplicated elective surgical procedure or over the age of 40 without additional risk factor who have minor elective abdominal surgery for less than 30 minutes. In the absence of prophylaxis, these patients have less than10% risk of developing proximal DVT and 0.01% risk of fatal pulmonary embolism.

Moderate risk patients are those over the age of 40 who have elective general abdominal surgery performed under general anaesthesia lasting for at least 30 minutes. Advancing age, presence of malignancy, prolonged bed rest, varicose veins and obesity, increases risk in this category. In the absence of prophylaxis, patients in this risk category have a 10-40% risk of DVT and 0.1 to 0.7% risk of PE.

High risk patients are those who have a recent DVT, who require surgery and those who undergo pelvic or abdominal surgery for advanced malignant disease or major orthopaedic surgery of the lower limbs. In the absence of prophylaxis, these patients have a 40-80% risk of DVT and 1 to 5% risk of fatal PE (Salzman and Hirsh, 1982).

The validity of an instrument is determined by its ability to measure what it claims to measure. Content validity is primarily concerned with whether the items of a healthcare assessment tool are relevant and adequate to the content area under investigation. Essentially, it involves seeking assurance that the items of the tool are very representative and comprehensive (Bowling, 1995). On the other hand, construct validity is the degree to which an instrument measures the theory or hypothesis under investigation. It is also the ability of the assessment tool to confirm the expected hypothesis

and discriminate between groups (Anthony, 1999). For example the DVT risk assessment tool should be able to discriminate between patients who develop DVT and those who do not. Each item was examined to see whether it was relevant and any unrelated items were discarded. The related items were drawn from extensive literature review and compelling evidence from research studies discussed in chapter two. The representation of all the clinically justified variables and evidence based items derived from theory underpinning the causation of DVT, are indicative to both the content and construct validity of the Autar DVT scale. Founded on the universally recognised Virchow's triad, the DVT scale has addressed all the relevant aspects of the phenomenon of interest. The biological basis of DVT in relation to the thrombogenic mechanism of the high risk factors constituting the scale is given in table 3.19.

Table 3.19: Thrombogenic mechanism of high risk factors in DVT

High risk factor	Vessel damage	Increased blood Coagulability	Stasis	Decreased Fibrinolysis
Increasing age			+	
Obesity			+	+
Immobilisation			+	
Oral contraceptive		+	+	+
Pregnancy and Puerperium		+	+	+
Leg trauma	+	+	+	
Surgery	+	+	+	+
Ulcerative colitis	+	+		
Sickle cell anaemia			+	
Haemolytic anaemia			+	
Polycythaemia		+	+	
Heart failure			+	
Myocardial infarction		+	+	
Malignancy	+	+	+	
Varicose veins	+		+	
CVA			+	
Previous DVT	+		+	+

+ denotes the manifestation of a thrombogenic mechanism

Data: Autar,1996 c.

Summary

The Autar DVT scale was developed as an assessment tool for predicting clients at risk of deep vein thrombosis. Based on Virchow's triad in the pathogenesis of DVT and culled from an extensive literature review and well-founded research findings, the DVT scale can claim high content and construct validity. The DVT scale comprises seven well-recognised categories of risk factors, namely: age specific group, mobility, build/ body mass index, special risk category, trauma, surgical intervention category and high-risk diseases. Each risk factor is assigned a risk score based on relative risk and the guiding principle of the DVT scale is that risk factors are additive.

One risk factor may be present in the low risk group, rising to 2-4 factors in the moderate risk group. More than 4 factors are present in the high risk group.

The scoring system identifies the patients into one of the four risk categories: no risk, low risk, moderate and high risk. A high-risk score indicates that the patients belong to a high-risk category and they have a 40-80 per cent risk of DVT and 1.5 per cent risk of fatal PE.

Chapter Four

Research methods

Research questions

The purpose of this quantitative and longitudinal study was to evaluate the Autar DVT risk assessment scale on orthopaedic, medical and surgical directorates. These diverse clinical areas were essentially targeted on account of the varying levels of risk by patient group (table 1.1), as the Autar DVT scale was designed to have universal application. Specifically, the research questions sought to assess the DVT scale in relation to its:

- consistency in the clinical assessment of patients.
- sensitivity, specificity and predictive validity.
- practical utility and application.

Available data were collated to determine current venous thromboprophylaxis strategies on the directorate, in the context of the recommendations of the consensus groups (NIH, 1986; ACCP, 1996; International Consensus Statement, 1997; THRiFT, 1998).

The study was undertaken prospectively and patients recruited were followed up for a minimum of three months, following discharge from the ward. In this predictive design data from three groups of patients were pooled to evaluate the DVT risk assessment scale, as a tool in the decision making process, for the venous thromboprophylaxis programme.

Ethical clearance and access negotiation

As required by the local Research Ethics Committee, a formal application was submitted for approval of the study and ethical clearance (appendix 2).

The UKCC code of professional conduct (1992) stipulates that "Each registered nurse, midwife and health visitor shall act, at all times in such a manner to:

"Safeguard and promote the interests of individual patients or clients".

No harm or inconvenience was foreseen to the patients by testing the reliability, validity and practical utility of the DVT scale. The risk calculator was a refinement of an assessment strategy and any new tool must be rigorously tested for its effectiveness, before it is allowed to enter the professional market for general use (Cormack & Reynolds, 1992). For this reason, the study was essentially kept to a prospective data generating project. The staff involved were advised not to alter the management of the patients in the light of the risk assessment, but to give the standard care normally given to patients. Any unusual attention to DVT risk might deliberately result in more preventative strategies with some consequential effect on evaluating the predictive validity of the DVT scale. The oral consent of the patients was taken by the data collectors. Patients recruited to the study were informed that data collected were being utilised to validate the DVT risk assessment tool. They were reassured that data collection would not in anyway alter their treatment and that information gathered would be very confidential.

Following approval and ethical clearance by the Research Ethics Committee overseeing the targeted NHS trusts (appendix 3), applications were submitted to the Research and Development units of each of the selected hospitals for indemnity of the study by the trusts (appendix 4). Access to the clinical areas was then negotiated. Written consents were obtained from the clinical medical directors of the three targeted directorates (appendices 5.1, 5.2 & 5.3).

At the same time, the three nurse managers of the clinical directorates were approached to negotiate the support of clinical nursing colleagues for participation in the data collection. In fact, the nursing staff in the targeted clinical areas were very keen to get involved and saw their participation in the study as consistent with their Post Registration Education and Practice (PREP) requirement for continuing professional development (UKCC,1994). The purpose of the PREP project was to develop standards for a framework of post registration education and practice which would contribute to the maintenance and development of professional knowledge and competence. The responses from the nursing staff were favourable as they stood to gain

professionally, in terms of development of their research awareness and profile and the implementation of evidence based practice.

Sample size and subjects

There are two main reasons to be concerned with sample size. First of all, the findings should be generalisable to the population that has been sampled. Secondly, the study should have a reasonable chance of detecting significant effects where they exist, that is, the study has adequate power. A study that has a large sample to be generalisable but which has a low probability of detecting effects, is of little value (Ingram, 1998).

Many factors interplayed in deciding the appropriate sample size for this study and the goal was to obtain a large enough sample to show statistical significance, yet be expedient and economical at the same time. Sample size is often a compromise between statistical precision and resource to hand. Increasing the sample size to the detriment of the quality of the rest of the research design may not be worthwhile (Jordan, Ong & Croft, 1998). Effect sizes as in this study are likely to be small in new areas of research enquiry (Cohen, 1977).

In a previous small scale study (Autar, 1994) the DVT risk assessment scale was developed through an action research process. Action research is a form of on-going self reflective enquiry that is being commonly applied in nursing practice to bring about positive changes (Webb, 1989).

The DVT scale (1994) was tried and tested on a trauma/ orthopaedic ward for its practicality, consistency and predictive validity. Seven days risk assessment data collected independently by paired registered nurses on each of the 21 patients recruited were analysed and the instrument achieved a consistency of 70 and 97 percentage agreement in the two small reliability studies respectively. There is a consensus among behavioural scientists that an average of 70 per cent is necessary for consistency, 80 per cent is adequate and 90 per cent good. (Hartmann, 1977, House et al, 1981). At a threshold score of 16, the DVT risk calculator also achieved 100% sensitivity and 81% specificity. Overall, a predicted accuracy of 83 % was recorded in relation to the correct classification of those at risk and

those who were not (Autar, 1994). Despite the promising results recorded, the nature of the small study does not allow for generalisation of findings and population representation. The establishment of validity is an ongoing process and any additional validity testing requires the publication of a second report (Norbeck, 1985). Further validation of the DVT scale was therefore warranted in larger patient populations, across diverse clinical specialities. Wasson et al (1985) in suggesting rules for clinical prediction, recommend that instruments be tested in more than one setting to eliminate unusual, practice-specific relationship between the predictor (DVT scale) and the outcome (end point DVT).

A convenience sample of 150 patients were therefore recruited from the orthopaedic/ trauma, medical and surgical directorates of two local NHS trusts. Fifty patients were studied from each of the three clinical specialities. DVT is a problem common to all the three clinical areas and literature review confirms that the target populations have been well characterised. Varying incidences of DVT had been reported in the three specialities (Table 4.1) and they would therefore provide a good testing ground for validating the DVT risk assessment tool.

Table 4.1: DVT incidence in trauma / orthopaedics, general medical and general surgery specialities

Specialities	DVT incidence Weighted mean	95% Confidence interval
Trauma/ Orthopaedics Hip fractures	45	41-48
General Medicine	17	10-24
General Surgery	25	24-26

Data: Grace, 1993
Hirsh & Hoak, 1996
International Consensus Statement, 1997

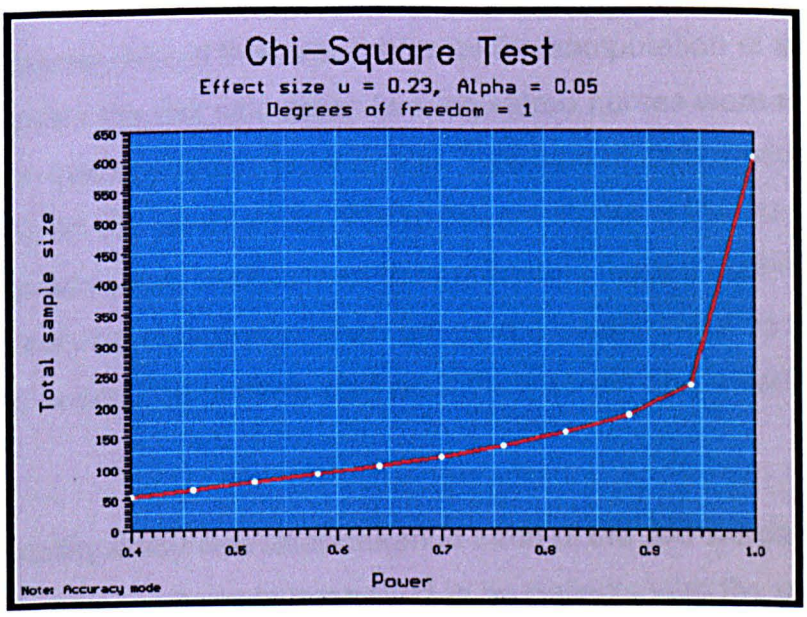
Calculation of sample size

In order to calculate the power and sample size, a priori analysis was undertaken, using the computer software G*Power programme and selecting Cramer's V statistic to determine the power of Chi Square for scores of patients with and without DVT. G*Power is a general power analysis programme, performing high precision statistical power analyses for most common statistical tests in behavioural research. It computes power value for given sample sizes, effect sizes and alpha levels as *a priori* power analyses as well as *post hoc* power analyses (Appendix 6).

Choosing the conventional alpha input of 0.05 and the minimum considered acceptable power (1-beta) of 0.80 as default, G*Power calculated a small to medium effect size of 0.23 (Table 4.2). A power of 0.80 implicitly means that a type II error (failing to detect an effect) is about one fourth as serious as type I error (falsely stating an effect, when no effect exists (Anthony, 1999). For the alpha input of 0.05 and given power of 0.80, the Cohen definition of large, medium and small (table 4.3) was used to calculate the sample size. A small to medium effect of 0.23 at a critical value of chi-square at 3.84 calculated a sample of 149 subjects (Erdfelder, Faul & Bucher, 1996).

Table 4.2:*A priori* calculation of sample size

Effect Size	**0.23**	**Lambda**	**7.8621**
Alpha	0.05	Critical value	Chi^2 (I)=3.8415
Power	0.80	Total sample size	149
Deg.fr.	1	Actual power	0.8017



Source: G*Power computer software.

Table 4.3: Cohen effect sizes (1988)

Effects: d	Sizes
0.10	Small
0.30	Medium
0.50	Large

The G*Power analysis was consistent with Cohen's effect size convention. Cohen (1988) claims that for new areas of research falling into this category, as in the validation of the DVT scale, group differences are likely to be small.

The available resources in terms of time, location and the willingness of the participants to co-operate primarily influenced the choice of population. Even though the only true representative sample would be one including the entire population (Knapp, 1996), there was a practical, ethical and economic limit in choosing the 150 patients, in addition to the recruitment of registered nurse participants for the data collection. In order to ensure that the information gathered on each patient was kept strictly confidential, it was felt that it would be more natural and practical for the own ward nurses to undertake the information gathering process. This would " protect all confidential information concerning patients and clients obtained in the course of professional practice" (UKCC,1992).

This strategy would then also allow for the computation of a kappa statistic to evaluate the risk calculator: two registered nurses were required to collect data on each patient independently. This type of data analysis also had a bearing on the sample size. Within the constraint of the number of registered nurses available as data collectors, kappa statistic estimation could only be undertaken when the paired nurses were on the same duty roster, noting that nursing staff work thirty seven and a half hours weekly shift.

A reliability study was undertaken in each of the five wards. The opportunity or convenience sample happened to be patients who the paired nurse assessors could get hold of, just like the sample available in a natural experiment. Convenience sampling is by no mean an ideal sampling method, since it is not a random process. However, in nursing research the predominant approach to sampling is non random sampling due to the type of population and the ethical considerations associated with them (Talbot, 1995). It would be wholly unethical and unacceptable to randomise an experimental and control group of subjects, in order to validate the DVT scale. There is compelling evidence on the efficacy of venous thromboprophylaxis and to withhold preventative strategies, for the sake of estimating the sensitivity and specificity of the DVT risk calculator, is an omission of duty of care and considered as clinical negligence (Parker-Williams & Vickers, 1989).

The clinical nature of nursing research therefore makes it difficult to use probability sampling and the majority of nursing studies continue to rely on sample of convenience (Polit & Sherman, 1990). While it is possible to randomise a sample from a list of a few patients coming in as planned admissions, it is not possible to do so for the bulk of admissions which are non elective and of emergency type. Besides, admission to the wards within a directorate takes place on a rotational daily to weekly basis (take days). Arguably, there were some elements of sample randomisation and generalisation as any patients admitted to that particular ward, on the so-called"on-take" days when the particular paired assessors were on duty, stood to be recruited to the study. This was believed to be important to the

integrity of the research design as all patients admitted on the specific days would be studied and therefore self-selection bias was reduced.

While acknowledging the limitations of a convenience sample, every effort was made to ensure that the sample was representative of the clinical population. The phenomena under investigation were fairly homogenous within the population at large, further minimising bias. One of the strengths of the study was that patients exhibited a wide variability of scores, ranging from the very highest score of 27 to the lower of 1 for the no risk category. Score variability was evidenced in the sample representation of the various risk categories (Table 4.4). The estimated mean risk assessment score of the sample was 10, a mean risk assessment score of 12 for patients with DVT and overall standard deviation of 5. A risk score range of 1-27 for the population sample was recorded.

Table 4.4: Sample representation of the risk categories.

Risk categories	Number of patients	%
High	19	13
Moderate	37	25
Low	50	34
No risk	42	28
Total	148	100

The homogeneity of the sample also added weight to the claim of generalisability. If the individual subjects are very much alike in all variables, other than the one being measured, a smaller sample suffices (Crookes & Davies, 1998). There was little variability in the characteristics of the population. The fifty subjects on the trauma /orthopaedic wards were prototypical of subjects with hip fractures, as cardiovascular disorders were to those on the medical wards (Claggett et al, 1992). Most of the patients on the surgical directorate had undergone major abdominal surgical intervention.

A small standard error of mean (SEM), referring to an estimate of the population parameter, also added further credence to the homogeneity of the population. With a SD of $\triangleq$ 5, the estimated SEM was $\triangleq$ 0.04 (appendix 7) The smaller the SEM, the more accurate are the sample means as

estimates of the population value (Campbell & Machin, 1993; Polit 1996). The SEM was much smaller than the SD because the sample mean was not as spread out as the original risk assessment scores.

The proportion of the population was estimated from the data pooled from the three clinical directorates. The following equation was applied to calculate the population percentage at 95 % Confidence Interval:

Equation $$1.96 \times \frac{PQ}{\sqrt{n}}$$

Where: 1.96 is a constant for 95% confidence limit.

P: proportion of the sample with DVT= 28/148=0.19.

Q: proportion of the sample without DVT= 120/148= 0.81

1.96 x $\sqrt{}$ 0.19 x 0.81/ 148

1.96 x $\sqrt{}$ 0.1539/148

1.96 x $\sqrt{}$ 0.0103

1.96 x 0.3224= 0.06.

0.06 added to the proportion of sample with DVT=0.06+0.19 = 0.25 (Upper CI).

For estimation of the lower limit of CI, 0.19 was subtracted from the upper limit of 0.25, to yield a CI = 6 to 25%.

The trade-off for the margin of error is influenced by sample size and the choice of confidence interval.

The larger the sample, the smaller is the margin of error or the width of interval and the converse holds. On the other hand, a higher confidence interval increases the width of the margin, resulting in less precision.

Incidence of DVT increases exponentially and sharply with age from 1 per 100,000 people per year in childhood to nearly one per cent in old age (Rosenthal, 1999). It was for this reason that adult clinical areas were targeted, ensuring that children were excluded, and that adult patients only recruited, irrespective of age groups and sexes. Sex as a predictor of DVT is

not a significant and independent factor (Coon et al 1973;Coon, 1978). Epidemiological data analysed by Nordstrum et al (1992) reveal no difference between men and women in relation to the incidence of DVT. Any gender-related risk would be on account of additional risk factors such as oral contraceptive or Hormone Replacement Therapy or Pregnancy and Puerperium.After taking into account the risk factors; it appears unlikely that sex difference exists in the incidence of DVT (OHE 1996).

Another inclusion criterion was that all patients recruited to the study had to be risk assessed within 24 hours of admission. The choice of 24 hours admission was considered timely for optimum predictive accuracy when patients were very dependent because of their self care deficit and were at greater risk of DVT due to the acute nature of their clinical condition or surgical intervention with accompanying fibrinolytic shutdown (Merli & Martinez, 1987; Kakkar & Stringer, 1990; Stewart Rose, 1998). Thrombi of clinically significant proportion are present as early as the first 24 hours of admission (Brown & Newman, 1995). In trauma and orthopaedic patients DVT occurs as early as preoperatively. Roberts et al (1990) reported a 9 % prevalence of preoperative DVT and Hefley et al (1996) recorded a DVT incidence of 62 per cent in those patients with hip fractures whose operations were delayed more than 48 hours.For this reason, DVT risk assessment should be undertaken as early as possible and for those patients who would be having surgery within the 24 hours deadline, they would be risk assessed immediately on their return from theatre. In the immediate postoperative period, both hypercoaguable state and stasis are at their maximum (Nicolaides, 1990). Consequently, patients are most vulnerable due to the disruption of the endothelial integrity arising from widespread vasodilatory effects of many general anaesthetic agents (Comerata et al, 1989).Once the endothelium of the veins is disrupted, thrombogenic subendothelial substances are exposed and stimulate clot formation (Merli & Martinez, 1987). The majority of thrombi formation occurs at the time of surgery or in the immediate postoperative period when hypercoagulability and venous stasis are at their maximum (Nicolaides, 1990). This is another reason for the choice of the 24 hours cutoff period for undertaking the DVT risk assessment procedure. Inevitably, some surgical

patients who were scheduled for surgery within 24 hours of admission, but had their procedure postponed, would not be considered for the study.

In order to ascertain that the 150 patients recruited would provide sufficient data for evaluating the predictive validity of the DVT scale and allow for the statistical analysis to be significant, all patients admitted primarily for treatment of acute DVT were excluded from the sample.

Finally, many patients discharged from hospital continue to be at risk, as risk factors persist in some of them. Besides, most methods of prophylaxis are applied to patients in hospital often for the peri-operative period and seldom beyond 7 days. The mobility of some patients recovering from the surgical procedure may also be decreased when they get home.
25% of patients who do not have DVT on the ward did so within six weeks of discharge in the community (Scurr et al, 1988; Scurr, 1990). This problem is further compounded by the limited healthcare resources, requiring earlier patient discharge. As a result, there is an inverse relationship between the duration of hospitalisation and the probability of developing DVT following discharge home (Hass, 1997). Consequently, all patients were followed up for a minimum of three months and monitored for occurrence of DVT. Of the 150 patients recruited to the study, it was not possible to follow up two patients post discharge and their data were excluded for the estimation of the sensitivity, specificity and predictive validity of the DVT scale.

Data collection tools

In order to evaluate the predictive validity, consistency, sensitivity and specificity of the DVT risk assessment scale, two data collection tools were constructed, piloted and developed. The Autar DVT risk assessment chart was at the primacy of the information-gathering process for evaluating the consistency and validity of the assessment tool. Adjunctively, a Likert type questionnaire measured the practical utility of the DVT scale.

The double- sided DVT risk assessment chart (figure 4) was prominently landscaped to exhibit the DVT scale on one side and a BMI ready reckoner on the reverse. Body build as a risk category can be very subjectively assessed.

Figure four

AUTAR DVT RISK ASSESSMENT SCALE

ADDRESSOGRAPH

Name:

Unit No:

Ward:

AGE SPECIFIC GROUP

Age Group	Score
10-30	0
31-40	1
41-50	2
51-60	3
61+	4

BUILD/BODY MASS INDEX (BMI)

Build	BMI	Score
Underweight	16-19	0
Average	20-25	1
Overweight	26-30	2
Obese	31-40	3
Very Obese	41+	4

$BMI = wt(kg)/ht(m)^2$

MOBILITY

Risks	Score
Ambulant	0
Limited (uses aids self)	1
Very Limited (needs help)	2
Chair bound	3
Complete Bed rest	4

SPECIAL RISK CATEGORY

Oral contraceptive:	Score
20-35 yrs	1
35+ yrs	2
Pregnancy/Puerperium	3

ASSESSMENT PROTOCOL

Score ≤6	**No risk**
Score 7-10	**Low Risk (<10%)**
Score 11-14	**Moderate Risk (11-40%)**
Score 15≥	**High Risk (>41%)**

THROMBOPROPHYLAXIS

Please tick the appropriate prescribed DVT prophylaxis

- **Leg Elevation** ☐
- **Graduated Elastic Compression Stockings (GECS)** ☐
- **Pneumatic Compression (PC)** ☐
  - **Intermittent or** ☐
  - **Continuous** ☐
- **Heparin** ☐
  - Heparin Sodium (standard/unfractionated)
  - Calciparine (heparin calcium)
  - Minihep (heparin sodium)
  - Minihep Calcium (heparin calcium)
  - Monoparin (heparin sodium)
  - Monoparin Calcium (heparin calcium)
  - Multiparin (heparin sodium)
  - Pump-Hep (heparin sodium)
  - Unihep (heparin sodium)
  - Uniparin (heparin sodium)
  - Uniparin Calcium (heparin calcium)
- **Low Molecular Weight Heparin** ☐
  - Fragmin (dalteparin sodium)
  - Clexane (enoxaparin heparin)
  - Innohep (tinzaparin)
  - Logiparin (tinzaparin)
- **Warfarin** ☐
- **Others, please specify:** ☐
- **No Prophylaxis** ☐

TRAUMA RISK CATEGORY

*Score item(s) **ONLY** preoperatively*	Score
Head	1
Chest	1
Spinal	2
Pelvic	3
Lower Limb	4

SURGICAL INTERVENTIONS

SCORE ONLY ONE APPROPRIATE ITEM	Score
Minor Surgery (<30 mins)	1
Major Surgery	2
Emergency Major Surgery	3
Thoracic	3
Abdominal	3
Urological	3
Neurosurgical	3
Orthopaedic	4

HIGH RISK DISEASES

	Score
Ulcerative Colitis	1
Anaemia: Sickle Cell	2
Polycythaemia	2
Haemolytic	2
Chronic Heart Disease	3
Myocardial Infarction	4
Malignancy	5
Varicose Veins	6
Previous DVT or CVA	7

SCORING (within 24 hours of admission)

Identify appropriate items, add and record score below

Assessor	Date	Score

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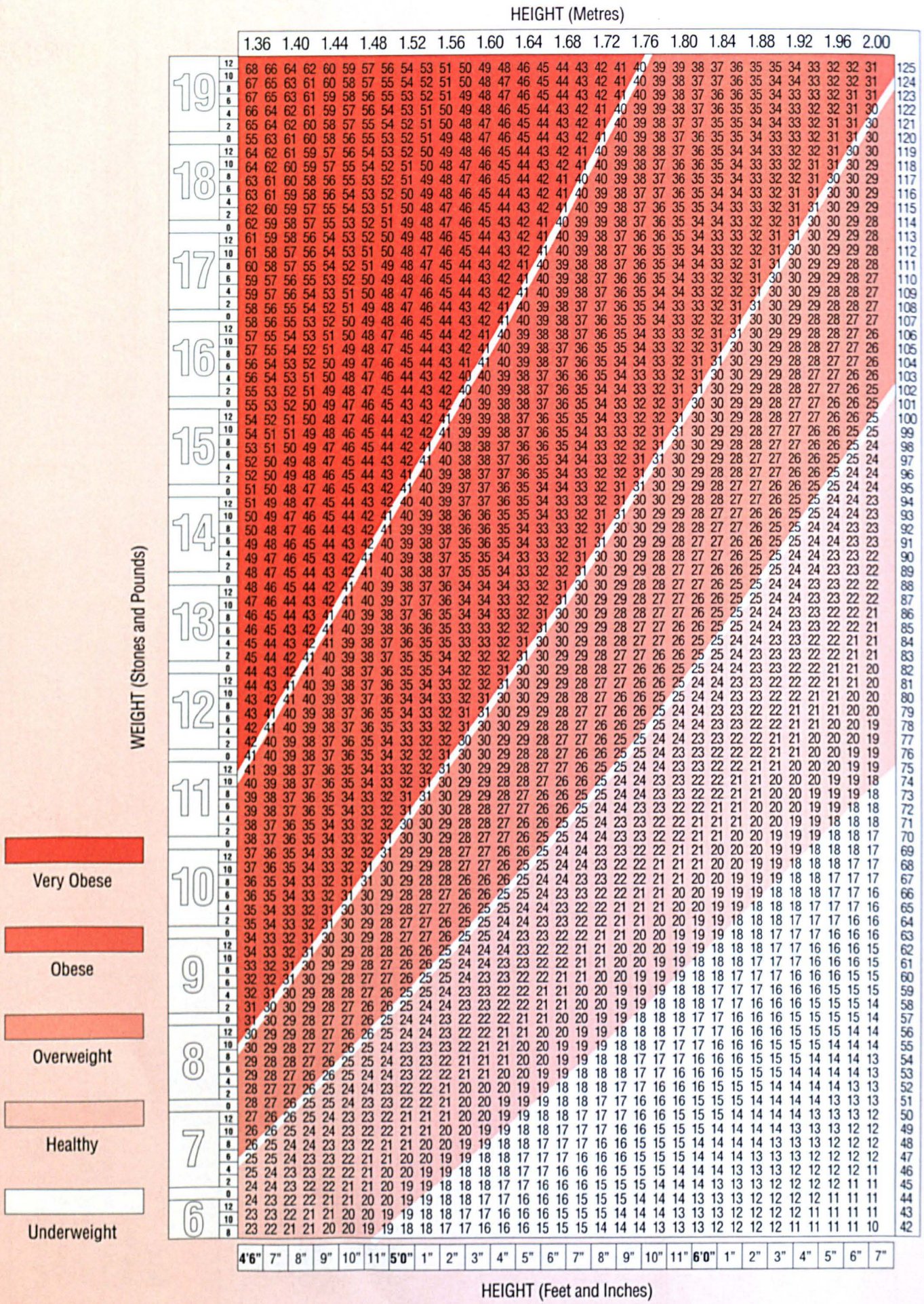
BODY MASS INDEX CHART



While a visual assessment can be a guide to whether or not a person is an appropriate weight or build, it is never an adequate or accurate measurement of a particular build. This is because different individuals and different cultures have different perception of what constitutes a particular build, an ideal weight, overweight or obese. What is an obese build can be construed as average by an obese rater as clinical judgement lacks a gold standard (Brennan & Hays, 1992). The BMI ready reckoner therefore facilitated assessors in objectively differentiating between the types of body builds and scoring this category of the DVT scale. Using commonly obtained data from a nursing health history, specifically the height and weight, one is able to calculate the Quetelet BMI. This is obtained by taking the weight in kilograms divided by the square of a person's height in meters. The BMI was supplementary to the clinical judgement of the raters. The lay-out of the DVT scale was designed by the researcher to facilitate the collection of the appropriate variables. However, the printed charts for undertaking the research study were financially supported and supplied by Huntleigh Healthcare (Luton) for acknowledgement of services provided in relation to DVT prophylaxis, with respect to the evaluation of the Flowtron Intermittent Pneumatic Compression (IPC) device.

The seven recognised categories of risk factors in the genesis of DVT were illustrated in distinctly bold boxed areas to assist the assessment process systematically and comprehensively. The addressograph on the top left-hand corner of the chart identified the patient by a predetermined code chosen by the assessors. The unit number of the patients, held on the Hospital Inpatient Data System, enabled access to the patients' case note library for tracing their final discharge destination and facilitated their follow up at the outpatient clinic, anticoagulant clinic or the GP surgery, whatever was appropriate.

All the pharmacological and mechanical venous thromboprophylaxis modalities were exhibited in the DVT chart. The pharmacological modality was illustrated by a comprehensive list of anticoagulants for primary prophylaxis extracted from the British National Formulary (BNF, 1994) and registered nurse assessors were required to tick what was actually prescribed. Such data would then be collated to audit the current DVT

prophylaxis protocol. For the trauma risk category, the registered nurse assessors were issued with specific instruction to score this subscale only pre operatively and this risk factor ceased to apply, once the therapeutic surgical intervention had been initiated to rectify the trauma or injury.

Further to supplement the instruction on the DVT risk assessment chart, a quick reference instruction card was constructed, outlining the systematic step by step completion of a DVT risk assessment. In brief, to complete the DVT risk assessment nurses were required to identify the appropriate risk factors, add and record the scores. The aggregate could then be referenced to the DVT assessment protocol to diagnose the individual risk category of the patient. The independently rated aggregate would also be computed by the researcher to estimate the kappa coefficient values.

Reynolds & Cormack (1992) propose certain criteria in the guise of the following five broad questions in the evaluation of a model for nursing:

1. To what extent does the instrument facilitate the identification of actual and potential health problems?
2. How does the tool enable a nursing diagnosis to be made?
3. Does the tool explain why patients respond to health problems in the way they do?
4. Does the tool enable clinicians to choose the appropriate nursing treatment required?
5. Does the tool provide an understanding of the desired outcome of nursing intervention?

Although the above questions were directed at the evaluation of a nursing model for clinical practice, the evaluation criteria are equally transferable and readily applicable to any nursing assessment tool.

It was within the broad framework advocated by Cormack & Reynolds (1992) that a postal questionnaire was constructed to evaluate the favourability and practical utility of the DVT scale. An attached cover letter explained the general purpose of the tool and freepost reply envelope enclosed to maximise response rate.

Postal questionnaire: Figure 5

Evaluation of the Autar DVT scale

Dear colleague,

Thank you for your support in the data collection process of the research project. Your views of the DVT scale are much valued and will be used to identify the strengths and areas in need of improvement.

Further to evaluate the practical application of the DVT scale, I am now asking for your help in answering the attached questionnaire.
For the sake of convenience, the questionnaire is divided into 2 sections. Section 1 deals with the biographical data, which are assured of confidentiality.

Section 2 addresses the general issues surrounding the practicality of the scale and the specific DVT risk categories.

On completion of the research project a copy of the report will be made available to your ward.

Thank you again for your help and please return the completed questionnaire in the stamp addressed envelope attached.

EVALUATION OF THE AUTAR DVT RISK ASSESSMENT SCALE

All information will be treated as confidential and no name disclosure is required.

SECTION ONE: BIOGRAPHICAL DATA.

1. How long have you been qualified?

2. What are your professional qualifications?

3. What is your job title \ clinical grade?

4. How long have you been working in this area of practice?

5. Have you worked in other clinical area(s) where the Autar DVT Scale is being used?

Please comment:

SECTION TWO: THE DVT SCALE

Please **tick** your response and make any relevant comments.

6. How many patients have you assessed with the DVT risk assessment scale?

less than 5	5-10	11-15	more than 15

7. The DVT scale enables me to:

7.1. Make a thorough DVT risk assessment based on the patient clinical profile.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

7.2. Individualise the nursing assessment of patients.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

7.3. Translate the risk factors as applicable to patients into DVT risk categories.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

7.4. State why patients are at risk.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

8. The DVT scale can be applied to facilitate care planning.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

9. The DVT scale **does not** enable me to take preventative nursing action to minimise risk

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

10. The DVT scale has no value as a teaching tool.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

11. The DVT scale has potential application as an audit tool for quality assurance.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

12. The DVT scale has practical application to my area of practice.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

13. The layout of the DVT scale facilitates the data collection process for risk assessment.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

14. The DVT scale enables me to complete a risk assessment within

Less than 3 mins	3-6 mins	7-10 mins	more than 10 mins

Any comments:

15 Please comment on the strengths of the DVT scale.

16. Please comment on the weaknesses \ limitations of the DVT scale.

17 Any comments on how the practical application of the DVT scale can be improved.

18. Age specific group:
This category is sufficiently clear to score.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

19. Mobility:

Scoring item from this category is reasonably clear.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

20. Build:

I am able to differentiate between underweight and average build.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Comments:

20.1. I am able to differentiate the overweight patients from the obese and very obese.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Comments:

21.Special risk category:

Please answer this question if it is applicable to your patients.

This category is reasonably clear for me to score.

Strongly Agree	Agree	Neutral	Strongly Disagree.

Any comments:

22. Trauma risk category:

Please answer this question if this category is applicable to your area of practice.

I am reasonably clear as to what constitutes scoring for lower limb injuries.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

Please answer this question if this category is applicable to your area of practice.

23. Surgical intervention category:

I am able to differentiate between minor and major surgery

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

23.1. I am able to differentiate between major surgery and the other types of surgery.

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

24. High risk diseases:

I have no difficulty scoring any item from this category:

Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree

Any comments:

The postal questionnaire (figure 5) was primarily preferred for its low cost data collection and processing and avoidance of interviewer bias. The researcher has a very good professional working relationship with the clinicians and this rapport could inadvertently influence the respondents towards a bias and socially desirable response. The postal questionnaire also made possible reaching the respondents who were dispersed across the various clinical settings of the University Hospital of Leicester NHS Trust. Another deciding factor for the choice of postal questionnaire over interviews was that at the time of access negotiation and staff preparation, concerns were raised by clinicians. While nursing staff would voluntarily and willingly assist with the study, their involvement should be kept to the very minimum. At a time when already chronic staff shortage generates increased nursing workload excessive staff time demands would cause additional stress and compromise the duty of care. There was consensus by the clinicians and the researcher that a scheduled interview would be inappropriate, more disruptive and inconvenient to the nurse participants, than a postal questionnaire which could be responded timely and at their convenience.

The postal questionnaire was designed to generate a mixture of:

- Quantitative data for simple descriptive statistical analysis
- Qualitative data to provide insights into the practical application of the DVT scale.

It was designed to maximise coverage of the pertinent issues, response rate, ease of administration, cost and time effectiveness (da Vaus, 1993). Although a large number of questions increase the reliability of an evaluation tool, the use of more than 30 items would test the respondents' patience (Cooligan, 1996).

The questionnaire was composed of 29 items conveniently split into two sections. In the organisation of the items, the funnel approach was chosen. Section one addressed the biographical data of the participating nursing staff, in relation to their professional experience and competence. This biographical data would be used as a basis for explaining similarities and differences in responses to the items in section two. In section two, the questions were narrowed down to the scope of more specific evaluation of

the practical application and clarity of the DVT risk calculator. Where appropriate, to reduce the complexity of the statement, some items as illustrated by statements 7 and 21, were broken down into logical and specific components, to deal with one task or concept at a time, and enabled respondents to interpret them in the same way. It also ensures that the investigator actually gets the answers to the questions which respondents are willing and able to give and be critical about them in a constructive way. Both closed and open questions were used judiciously, so that the strength of one offsets the weakness of the other (Parahoo, 1997). The questions themselves were short and arranged in a logical sequence, easing the respondent into the more important and complex issues further on.

A Likert-type scaling procedure was applied to section two of the tool. Respondents were required to place themselves on one of the five points degree of agreement on an attitude continuum for each statement, running from Strongly Agree to Strongly Disagree. The five points were given simple weights of 5,4,3,2,1 for scoring purposes but items 9 and 10 of the questions were reversed in negative mode and scored 1 for Strongly Agree and up to five for Strongly Disagree. This deliberate reversed item construction was designed to stop respondents filling in the scale automatic and carelessly by going in one column. Generally, there is a tendency to agree rather than disagree, resulting in response acquiescence set whereby respondents agree with the item, irrespective of the contents rather than disagree (Oppenheim, 1992). Items 15 to 17 were also deliberately constructed as open-ended questions to break the automatic response mode. The respondents would feel less frustrated with this lack of constraint, making the whole approach realistic. Respondents "rarely have to just agree and disagree without comments" Cooligan, 1996:p 87).

Odd items as illustrated in statements 8 and 9 were arranged in an unpredictable mixture of positive and negative statements. Such a format has the culminative effect of keeping the respondents actively thinking when making their response (Burns, 2000).

Items 18 to 24 were very specific to the seven risk categories of the DVT scale, and respondents were asked to comment on the ease with which they

chose the most appropriate risk factor for each of the subscales. Such responses would be analysed to establish the clarity and consistency of DVT risk assessment.
Additionally, for each item in section two of the tool, respondents were offered the opportunity to make general comments to obtain unanticipated answers, thus allowing for the collection of qualitative data collection. The respondents might also describe more closely their real views and increase the validity of the measurement (Fowler 1993).

At the launch of the staff preparation for the study nursing staff were briefed that a postal questionnaire would be used to evaluate the practical application of the DVT scale, It was not posted to them until data collection, using the DVT scale, was completed and the targeted number of patients recruited to the study. This delaying tactic ensured that the questionnaire was not misplaced and enabled the respondents to be fully familiar with the risk assessment tool, thus facilitating them making constructive feedback in terms of its strengths, weaknesses and practicality. Such feedback would then be used to modify and develop the DVT scale further.

The questionnaire was applied in its entirety across the three clinical directorates and respondents instructed to overlook items that were not relevant to their particular speciality. The trauma risk subscale was essentially applicable to the trauma/orthopaedic unit and the Surgical Intervention Category not applicable to the patients on the medical directorate.

All participants were assured that information gathered would be protected in strictest confidence. Name disclosure was not essential as a discreet numerical code identified the respondents.The reason was twofold. First of all, the coding enabled the researcher to monitor the response rate and that any non or slow response could be followed up by a polite reminder and sending out a second or third questionnaire as required. A 30% response rate is not unusual for mailed questionnaire (Waltz et al, 1991) and 60% is considered adequate (Talbot, 1995). A high response rate of 79 per cent was achieved (22/28) as illustrated in the response table below:

Table 4.5: Response rate of mailed questionnaire.

Speciality	No. of mailed questionnaires	No. of responses
Orthopaedics / Trauma	13	12
General Medicine	13	8
General surgery	2	2
Total	**28**	**22 (79%)**

N= 28

Secondly, while maintaining confidentiality, in the sense that only the researcher would know the identity of the respondents, the biographical data in section one of the questionnaire, in terms of their clinical and professional experiences, could be analysed to explain their qualitative responses.

Staff preparation

Following successful access negotiation and recruitment of registered nursing staff as data collectors, a full programme of staff preparation was undertaken. On each of the selected wards, several half-day workshops were held. They were all scheduled at the convenience of the staff, so that all that participated in the project had uniform briefing and training on data collection with the DVT scale. This was judged to be fundamental to the accuracy of data collection and the evaluation of the inter rater reliability of the instruments in relation to its measure of equivalence and consistency.

Inter rater or inter observer reliability refers to "two or more trained observers or raters making the same observation simultaneously and independently recording the relevant variable, according to a predetermined plan" (Polit & Hungler, 1985:124).

Further to the evaluation of an instrument, Washington and Moss (1988) summarised six pragmatic aspects of the process of establishing interrater reliability, namely:

- Understanding of the theoretical perspective.
- Familiarisation with the research instrument.
- Number of subjects.
- Specific time frame.

- Concerns for interfering variables.
- Scoring and discussion.

All the six aspects proposed by Washington and Moss (1988) were applied in the preparation of the staff for the study.

Understanding of the theoretical perspective

A comprehension of the theoretical framework underpinnings the DVT risk calculator was given to the staff who engaged in the study as an essential beginning point. A collection of all published articles in relation to the DVT scale was made available to them. The rationale for each of the risk categories of the DVT scale was explained and the constructs underlying the items were fully explained. If there was a question about the interpretation of a particular item, the theoretical construct served as an important point of reference to clarify the item. Such discussions provided a foundation for post observation sessions when raters compared their independent ratings.

Familiarisation with the research instrument

Familiarisation with a research instrument promotes consistency in its application. The study focused on the refinement of a risk assessment tool and because it was new, training was imperative. The staff were orientated to the risk assessment factors and the scoring of the DVT scale. A trial run was rehearsed: those who engaged in the study tried out the DVT risk assessment scale on a few patients and any matter arising discussed and clarified. It was important that the instrument had clear instruction and that each item was stated unambiguously so that the item was interpreted only one way. Ambiguity of item increases error and variance and clarification enhances correctness of interpretation (Kerlinger, 1986).

Number of subjects

In the reliability studies, fifty patients were targeted from each of the three clinical directorates. The criteria for inclusion of subjects were explained and staff instructed to obtain oral consent from those recruited. Specific criteria included such as special risk categories, trauma risk factors, surgical intervention and high-risk diseases were also fully explained.

Specific time frame

To ensure that the same set of variables were rated a beginning time and ending time was determined. Risk assessment was to be undertaken within 24 hours of admission to the ward. In the case of patients scheduled for surgery within the 24 hours defined time frame, assessors were instructed to risk assess the surgical patients on return from theatre recovery, when they were deemed to be at optimum risk. By limiting the time frame, the raters' recall of events is enhanced and accuracy of scoring increased. This strategy allows for an encapsulated set of data of the subjects within the specified time to be observed and rated, using only the relevant items of the instrument.

Interfering variables

Interfering variables may influence the raters' performance and accuracy. The Trauma Risk Category, which applies only preoperatively, was concurrently scored postoperatively. The trauma risk category ceases to apply after surgical intervention and this discrepancy encountered in the dry run was clarified and problem resolved.

Scoring and discussion

Further to ensure that the scoring procedure was carried out independently, the investigator was available for supervision of practice and consultation. Data collected were kept in separate two box files, with each rater provided with one. This strategy ensured discreteness, confidentiality and independent rating of variables. The scoring was collated by the investigator and items of agreement and disagreement were evaluated and fed back to the raters.

Procedure

In order to maintain the integrity of the kappa statistic, all the staff were given precisely the same briefing at the DVT workshop to enable them to share a common frame of reference. Additionally, the registered nurse assessors were paired for data recording by virtue of equality of clinical

experience so that parity and consistency of data recording could be achieved.

The following step by step process was pursued in the risk assessment and data collection process:

It was left to the discretion and convenience of the paired assessors to decide when they should simultaneously and independently risk assess the patient within the 24 hours deadline. Patients undergoing surgery within the 24 defined time frame, would be risk assessed on return from theatre. Each patient would have two completed risk assessment charts by the end of the procedure.

As the data was leaving the clinical areas for analysis by the researcher, to conceal the identity of the patients a coding system was applied. Only the data collectors and the researcher would be able to identify the subjects. Before the assessment process, the paired assessors would choose a predetermined numerical code for the patient from the coding sheet supplied. For instance, the first patient would bear the numeral code of W100/01 followed by W100/02 for the second subjects and so on. Each code was to be used once and recorded in the addressograph section of the chart. The assessors would also record the patient name against the chosen code on the coding sheet, to enable the researcher to follow up patients on discharge.

To risk assess the patient, the paired assessors simply ring out the scores in the following subscales:

Age specific group: This applied to all patients and the assessors would score the appropriate age group.
Body Mass Index (BMI): This applied to all patients and again the assessors would ring out the appropriate item. The BMI ready reckoner on the reverse of the DVT chart provided a quick referencing guide to the raters.

Mobility: This subscale also applied to all patients and the appropriate level of mobility recorded.

Special risk category: This box was scored only if applicable to patients.

Trauma risk category: This category applied selectively to trauma patients. One or more items might be applicable but only preoperatively. This category ceased to apply when the patient had undergone surgery to repair the trauma.

Surgical intervention category: The raters had to score only one most appropriate item form this category.

High risk diseases: This category would be scored as applicable to the patients. In some subjects more than one item would be scored as applicable.

Venous thromboprophylaxis: In the initial assessment, the assessors would complete this box. The investigator would record any subsequent thromboprophylaxis and this helped to differentiate between primary and secondary prophylaxis.

To calculate the assessed patient overall score, the raters would simply add the ring- out scores. Aggregate score recorded placed the patients into one of the risk categories.

Finally, the independently risk assessed charts kept in a box file would be collated by the researcher for measure of equivalence. Any discrepancy or disagreement between the raters would be discussed with them.

This standard procedure was implemented in all the three clinical directorates of the NHS trusts. Strategically, one trust was targeted at a time and this freed the investigator to concentrate in one area of practice and manage the research process effectively. The investigator's decision to target one NHS trust at a time was influenced by several pragmatic factors. The rationale for singling out one NHS trust was as follows:

- It enabled the researcher to make more frequent visits to the clinical research area, which would not have been possible if the study was being undertaken simultaneously on multi-sites of the trusts. As a result, the limited human resource was channelled effectively.

- It enabled the research process to be managed strictly within the approved protocol.
- It facilitated prompt feedback to staff on queries relating to the data collection process and thus prevented departure from the agreed protocol.
- Focusing on one clinical area enabled the researcher to submit up to date and reliable reports to the research and development office of the trust as required within the terms of indemnity of the project.
- It facilitated close monitoring of the patients for the predicted outcomes.
- It enabled for a group of 50 patients to be followed up after discharge, noting that liaison with general practice, anticoagulant clinics, outpatient clinics, case notes library and community nursing services was paramount for monitoring predicted outcomes.

The data derived from a snapshot of 150 patients would be subjected to the following statistical analyses to evaluate the effectiveness of the DVT scale:

- kappa statistic to measure the inter rater reliability of the DVT scale.

- Bayesian method to calculate the sensitivity, specificity, predictive validity and likelihood ratios of the tool. Bayes' theorem links the probabilities of events before an experiment (*a priori*) to the probabilities of events after an experiment (*a posteriori*) Campbell, 2001).

- A Receiver Operating Characteristic (ROC) curve to determine the best cutoff score for optimal predictive accuracy.

- Logistic regression to evaluate the discriminatory power of the independent variable in the causation of DVT.

- A postal survey to evaluate the practical application of the DVT scale.

Finally, data from all sources would also be pooled and analysed to determine the current venous thromboprophylaxis protocol in relation to the recommendations of the consensus groups.

Summary

A longitudinal study was undertaken to evaluate the DVT risk assessment for its consistency, predictive validity, sensitivity, specificity and practical application. Available data was also analysed to determine how current venous thromboprophylaxis strategies compare with the recommendations of national and international consensus groups. A convenience sample of 150 patients were recruited from the orthopaedic, medical and surgical directorates of two trusts.

Five reliability studies were undertaken to estimate the consistency of the DVT scale. Registered nurses were paired in terms of parity clinical knowledge and experience and were required to risk assess patients independently with the risk assessment tool, within 24 hours of admission. The independent recordings were subjected to kappa statistics to measure the inter rater reliability of the DVT scale.

To evaluate the predictive validity of the instrument, the predicted patients were monitored for any DVT occurrence during their hospital stay. They were also followed up for a period of three months post discharge as DVT is a continuing problem and 25 per cent of patients develop DVT after discharge home.

At the end of data collection on each ward, users (registered nurse assessors) of the DVT risk assessment scale were sent a postal questionnaire (figure 5) to evaluate the overall effectiveness and practical application of the DVT scale.

Chapter Five

Results

The purpose of this chapter is to analyse data gathered, in terms of the aims of the study outlined in chapter one. Data collected and pooled from the 150 patients were analysed to evaluate the consistency, predictive accuracy, sensitivity and specificity of the DVT risk assessment scale. An instrument may have considerable ability to discriminate yet being of little value for patient management if it does not have practical application (Zweig & Campbell, 1993). A practical tool that is an optimal risk predictor must also be widely available, standardised, reproducible, cheap, user friendly and well defined (Lowe, 1993). Postal questionnaires were scrutinised to determine the practical application of the DVT scale. Available data on venous thromboprophylaxis were also analysed to establish a current local DVT prophylaxis protocol, in relation to the national and international consensus groups recommendation on venous thromboprophylaxis.

Reliability of the Autar DVT scale

Reliability is the first characteristic that any tool must possess and this is an absolute precondition to validity (Gibbon, 1995). Reliability can be conceptualised in terms of stability, equivalence and consistency (Oppenheim 1992). It is a measure of the stability of an instrument in relation to the repeatability of the data captured (Anthony, 1999).

The question of whether a tool is reliable or repeatable is therefore a significant consideration in assessing a screening tool such as the DVT predictive index. After all, clinical judgements lack the gold standard (Brennan & Hayes, 1992). Agreement is the proportion of all patients about whom the observers agree on the presence or absence of an observation or event. Agreement of two or more observers is defined as inter-observer agreement and agreement of an observer with themselves regarding repeated observations is termed intra-observer agreement (Koran, 1975). Inter rater reliability is the consistency of performance between two or more assessors, in relation to the degree of agreement in rating when carried out independently but simultaneously on the same person (Tomalin et al, 1992).

Estimates of reliability computed by different procedures for the same instrument are not identical (Polit, 1996).

Hartmann (1977) demonstrated that percentage agreement and correlation like procedures such as kappa (Cohen, 1960) often yield very different values when used for the same data. The reason for this difference is that a correlation like estimate of 0.00 indicates randomly occurring agreement while a percentage agreement of 00 implies absolutely no agreement (Lewin & Wakefield, 1979).

In order to communicate variability in the data captured for the establishment of the consistency of the DVT scale, both total percentage agreement (T%) and kappa statistic were reported. Percentage agreement is relatively simple to calculate more accurately. However, one of the major criticisms of percentage agreement is that it does not take into account the varying contribution of chance agreements to observed agreement rate. Some agreement between raters can generally be accounted for by chance alone, particularly if the variability among subjects is low and the number of categories limited.

It is for this reason that data pooled were also analysed to determine a kappa estimate and adjust for chance agreement. It is arguably an improvement on alternative methods of estimation for dichotomous nominal and categorical data (Fleiss, 1981). kappa estimation is now the most frequently used reliability measure for instruments providing categorical data (Soeken & Prescott, 1986). Also a clear understanding of reliability obtained in one study necessitates the reporting of more than one indication of reliability (Brennan & Hayes, 1992).
In order to calculate total percentage agreement (T%) the following equation was applied:

$$\frac{\text{Number of agreement}}{\text{Number of agreement \& disagreement}} \times 100 = (T\%)$$

kappa statistics (Cohen, 1960) were devised as an omnibus index of agreement for nominal scales that is, categories that lack a natural ordering. The advantage of the k coefficient over percentage agreement is its correction or adjustment for the amount of agreement expected to occur by chance. This apparent virtue of the kappa statistic has made it increasingly popular in studies of observer variability but has a problem of its own. Unweighted kappa treats all disagreements, large or small as equally serious and identical. Although weighted kappa (Cohen, 1968) addresses this weakness, it allows weight to be arbitrary in relative magnitude. This means that the magnitude of weighted kappa may be arbitrary and that standard weight should be used (Maclure and Willett, 1987).
kappa is symbolically expressed as:

$$K = \frac{Po - Pe}{1 - Pe}$$

Where: **Po** is the observed proportion of agreement
Pe is the chance expected proportion of agreement
The numerator Po-Pe is the proportion of observer agreement, explicitly corrected for the proportion of chance or expected agreements
The denominator **1- Pe** is similarly correction for chance agreement.

One of the striking paradoxes of this equation is that despite a relatively high result for interobserver agreement, the corresponding value of k can be relatively low. The value of k is the correction factor for chance agreement and is subtracted from the observed agreement and from perfect agreement. Consequently, k gets its highest value when Pe is as small as possible. On the other hand if Pe is large, the correction process converts a relatively high k to a low value of k (Feinstein and Cicchetti, 1990).
Another factor that affects k is the true prevalence of a diagnosis. If the prevalence of a diagnosis is high, the proportion of agreement expected by chance (Pe) is increased. This lowers the value of k (Byrt et al, 1993). Devising a prevalence index (PI), Byrt et al (1993) estimate the true prevalence of a diagnosis as (*a-d/N*) where *a* is the number with the condition minus *d* those without it, divided by the total. The prevalence index

can take values from -1 to +1. In the application of this index (28-120/148) a PI of -0.6 for DVT was recorded.

DVT is a universal problem that crosses all traditional departments, specialities and subspecialities of clinical practice (Bell and Simon, 1982). The Autar DVT scale was designed to have universal application to clinical practice. It was therefore fitting that a reliability study was undertaken in each of the five wards to evaluate the consistency of the instrument across diverse specialities.

kappa is appropriate for either dichotomous or polychotomous ratings and has been extended to 6 raters and 5 categories with a sample of 30 (Fleiss, 1971). But for the purpose of this study, pairwise kappa was applied.

There are three assumptions to be met when undertaking the kappa statistics. First of all, the subjects to be rated are independent of one another. Secondly, the raters operate independently of one another. The third assumption is that the categories are mutually exclusive and exhaustive (Cohen, 1960). In the typically reliability study, the raters were paired on the basis of equality of nursing experience and education, additional to having attended the same staff preparation brief. They were *a priori* deemed equal in their ability to make judgement and recording. The data collected simultaneously and independently by the paired assessors (Tables 5.1 to 5.5) were computed to calculate both the total percentage agreement and the kappa values.

Each table is then converted into a schema for examining variation between the two assessors on each of the five clinical areas. Recordings of assessor A are cross tabulated against the recordings of assessor B.

Table 5.1: Reliability study on trauma / orthopaedic ward C

Assessor A – B	Patients	Age	Build	Mobility	Special risk	Trauma	Surgery	High Risk Disease	Assesor A	Assesor B
									Total Scores	
	1	0-0	1-1	0-0	0-0	0-0	1-1	0-0	2	2
	2	4-4	1-1	4-4	0-0	4-4	0-0	0-0	13	13
	3	4-4	1-1	4-4	0-0	4-4	0-0	0-0	13	13
	4	4-4	0-0	4-4	0-0	4-4	0-0	0-0	12	12
	5	1-1	1-1	4-4	0-0	0-0	4-4	0-0	10	10
	6	3-3	2-2	0-0	0-0	0-0	4-4	2-2	11	11
	7	4-4	**0-1**	2-2	0-0	4-4	0-0	0-0	**10**	**11**
	8	1-1	1-1	4-4	0-0	4-4	0-0	0-0	10	10
	9	4-4	1-1	4-4	0-0	4-4	0-0	0-0	13	13
	10	2-2	1-1	0-0	0-0	0-0	4-4	0-0	7	7
	11	1-1	1-1	0-0	0-0	0-0	4-4	0-0	6	6
	12	4-4	0-0	4-4	0-0	4-4	0-0	0-0	12	12
	13	1-1	1-1	0-0	0-0	0-0	4-4	0-0	6	6
	14	1-1	1-1	0-0	0-0	0-0	4-4	0-0	6	6
	15	0-0	1-1	4-4	0-0	4-4	0-0	0-0	9	9
DVT	16	4-4	1-1	4-4	0-0	4-4	0-0	7-7	20	20
	17	2-2	1-1	0-0	0-0	0-0	**1-0**	0-0	**4**	**3**
	18	1-1	1-1	0-0	0-0	**0-4**	**4-0**	0-0	6	6
	19	0-0	1-1	**0-1**	0-0	0-0	4-4	0-0	5	5
	20	3-3	1-1	3-3	0-0	0-0	4-4	0-0	11	11
DVT	21	4-4	4-4	4-4	0-0	4-4	0-0	0-0	16	16
DVT	22	4-4	**1-0**	4-4	0-0	9-9	0-0	8-8	**26**	**25**
	23	0-0	1-1	0-0	0-0	4-4	0-0	0-0	5	5
	24	4-4	1-1	**0-1**	0-0	0-0	1-1	11-11	**17**	**18**
	25	0-0	1-1	0-0	0-0	0-0	1-1	0-0	2	2
	26	2-2	1-1	0-0	0-0	4-4	0-0	0-0	7	7

Total Percentage Agreement: $\frac{22}{26} \times 100 = 84.6\%$

Table 5.2: Agreement matrix on trauma / orthopaedic ward C

	No risk	Low risk	Moderate Risk	High risk	Total
No risk	9	-	-	-	9
Low risk	-	5	-	-	5
Moderate risk	-	1	7	-	8
High risk	-	-	-	4	4
Total	9	6	7	4	26

Although only 22 items of agreement were recorded out of the 26 ratings, converting them into four nominal categories of risk yielded 25 observed agreements for kappa correlation.

$$\text{Observed Agreement (Po)} = \frac{25}{26}$$

$$\text{Expected Agreement : (Pe)} = \frac{9\times9}{26} + \frac{6\times5}{26} + \frac{8\times7}{26} + \frac{4\times4}{26}$$

$$\frac{3.1+1.2+2.2+0.6}{26} = \frac{7.1}{26} = 0.27$$

$$K = \frac{0.96 - 0.27}{1 - 0.27} = \frac{0.69}{0.73} = 0.95$$

kappa value on ward C= 0.95

Table 5.3: Reliability study on the trauma / orthopaedic ward D

Assessor A- B	Patients	Age	Build	Mobility	Special risk	Trauma	Surgery	High Risk Disease	Assesor A	Assesor B
									Total Scores	
	1	4-4	0-0	0-0	0-0	4-4	0-0	0-0	12	12
	2	4-4	3-3	4-4	0-0	4-4	0-0	12-12	27	27
	3	4-4	0-0	2-2	0-0	0-0	0-0	5-5	11	11
	4	4-4	1-1	1-1	0-0	3-3	0-0	- -	9	9
DVT	5	4-4	1-1	0-0	0-0	4-4	0-0	0-0	9	9
	6	4-4	2-2	4-4	0-0	4-4	0-0	5-5	19	19
	7	4-4	1-1	4-4	0-0	0-0	2-2	0-0	11	11
	8	0-0	1-1	4-4	1-1	0-0	0-0	0-0	6	6
	9	4-4	3-3	4-4	0-0	4-4	0-0	0-0	15	15
	10	3-3	1-1	0-0	0-0	4-4	0-0	0-0	8	8
	11	1-1	1-1	0-0	0-0	4-4	0-0	0-0	6	6
	12	4-4	0-0	1-1	0-0	4-4	0-0	0-0	9	9
	13	**4-0**	**1-0**	**2-0**	**0-0**	**0-0**	**4-0**	**0-0**	**11**	0
	14	4-4	**1-2**	4-4	0-0	4-4	0-0	**7-10**	20	24
	15	4-4	1-1	**0-1**	0-0	0-0	**4-0**	0-0	**9**	**6**
	16	4-4	1-1	4-4	0-0	4-4	0-0	3-3	16	16
	17	4-4	1-1	4-4	0-0	4-4	**0-0**	7-7	20	20
	18	4-4	**1-2**	4-4	0-0	4-4	**0-0**	0-0	**13**	**14**
	19	4-4	1-1	4-4	0-0	4-4	0-0	0-0	13	13
DVT	20	4-4	2-2	4-4	0-0	4-4	0-0	0-0	14	14
DVT	21	4-4	3-3	4-4	0-0	4-4	0-0	0-0	15	15
	22	4-4	0-0	4-4	0-0	4-4	0-0	0-0	12	12
DVT	23	4-4	1-1	4-4-	0-0	4-4	0-0	0-0	13	13
DVT	24	4-4	1-1	4-4	0-0	4-4	0-0	0-0	13	13

Total Percentage Agreement: $\frac{21}{23} \times 100 = 91\%$

Table 5.4: Agreement matrix on orthopaedic ward D

B / A	No risk	Low risk	Moderate risk	High risk	Total
No risk	2	-	-	-	2
Low risk	-	4	-	-	4
Moderate risk	-	1	9	-	10
High risk	-	-	-	7	7
Total	2	5	9	7	23

Number in total= 23

One recording excluded from data analysis due to incompleteness of data.

Data Analysis: n = 23

Observed Agreement : $\frac{22}{23}$=0.96

Expected Agreement : $(Pe) = \frac{2\times 2}{23} + \frac{5\times 4}{23} + \frac{9\times 9}{23} + \frac{7\times 7}{23} = \frac{4}{23} + \frac{20}{23} + \frac{81}{23} + \frac{49}{23}$

$$\frac{0.26 + 0.86 + 3.5 + 2.1}{23} = \frac{6.63}{23} = 0.29$$

$$K = \frac{0.96 - 0.29}{1 - 0.29} = \frac{0.67}{0.71} = 0.94$$

kappa value on ward D is: 0.94

Table 5.5: Reliability study on medical ward 29.

Assesor A-B	Patients	Age	Build	Mobility	Special Risk	Trauma	Surgery	High Risk Diseases	Assessor A	Assessor B
									Total score	
	1	3-2	1-1	0-0	0-0	0-0	0-0	3-3	**7**	**6**
DVT	2	2-2	3-3	1-1	0-0	0-0	0-0	0-0	6	6
	3	4-4	1-1	0-0	0-0	0-0	0-0	5-5	10	10
	4	4-4	0-0	0-0	0-0	0-0	0-0	5-5	9	9
	5	4-4	1-1	1-1	0-0	0-0	0-0	4-4	10	10
	6	4-4	0-0	0-0	0-0	0-0	0-0	0-0	4	4
	7	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
DVT	8	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
	9	0-0	1-1	0-0	0-0	0-0	0-0	0-0	1	1
	10	4-4	1-1	1-1	0-0	0-0	0-0	0-0	6	6
	11	4-4	1-1	4-4	0-0	0-0	0-0	7-7	16	16
	12	2-2	0-0	4-4	0-0	0-0	0-0	5-5	11	11
	13	4-4	1-1	1-1	0-0	0-0	0-0	0-0	6	6
	14	4-4	0-0	1-1	0-0	0-0	0-0	0-0	5	5
	15	4-4	0-0	2-2	0-0	0-0	0-0	4-4	10	10
	16	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
DVT	17	4-4	2-2	2-2	0-0	0-0	1-1	7-7	16	16
	18	4-4	0-1	0-0	0-0	0-0	0-0	0-0	**4**	**5**
	19	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
	20	3-3	1-1	0-0	0-0	0-0	0-0	0-0	4	4
	21	4-4	2-2	2-2	0-0	0-0	0-0	7-7	15	15
	22	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
DVT	23	4-4	2-2	2-2	0-0	0-0	0-0	0-0	8	8
DVT	24	4-4	1-1	1-1	0-0	0-0	0-0	7-7	13	13
DVT	25	4-4	1-1	4-4	0-0	0-0	3-3	0-0	12	12

Percentage agreement:

$$\frac{23}{25} \times 100 = 92\%$$

Table 5.6: Agreement matrix on ward 29

B / A	No risk	Low risk	Moderate risk	High risk	Total
No risk	13	1	-	-	14
Low risk	-	6	-	-	6
Moderate risk	-	-	3	-	3
High risk	-	-	-	2	2
Total	13	7	3	2	25

Observed agreement: 24

Expected

agreement:

$$\frac{13\times14}{25}+\frac{7\times6}{25}+\frac{3\times3}{25}+\frac{2\times2}{25}=\frac{7.28}{25}+\frac{1.68}{25}+\frac{0.36}{25}+\frac{0.16}{25}=\frac{9.48}{25}=0.38$$

$$K=\frac{0.96-0.38}{1-0.38}=\frac{0.58}{0.62}=0.94$$

kappa value on ward 29 is 0.94.

Table 5.7: Reliability study: medical ward 30:

Assessor A – B	Patients	Age	Build	Mobility	Special Risk	Trauma	Surgery	High Risk Disease	Assessor A	Assessor B
									Total Score	
DVT	1	3-3	1-1	0-0	0-0	0-0	0-0	4-4	8	8
	2	3-3	1-1	0-0	0-0	0-0	0-0	0-0	4	4
	3	4-4	1-1	1-1	0-0	0-0	0-0	7-7	13	13
DVT	4	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
	5	4-4	2-2	2-2	0-0	0-0	0-0	0-0	8	8
	6	4-4	1-1	0-0	0-0	0-0	0-0	3-3	8	8
	7	4-4	1-1	1-1	0-0	0-0	0-0	3-3	9	9
	8	2-2	1-1	1-1	0-0	0-0	0-0	4-4	8	8
	9	3-3	1-1	0-0	0-0	0-0	0-0	0-0	4	4
	10	1-1	1-1	0-0	3-3	0-0	0-0	0-0	5	5
	11	4-4	1-1	4-4	0-0	0-0	0-0	5-5	14	14
	12	4-4	1-1	2-2	0-0	0-0	0-0	3-3	10	10
	13	4-4	1-1	**2-1**	0-0	0-0	0-0	0-0	**7**	**6**
DVT	14	3-3	3-3	**2-1**	0-0	0-0	0-0	3-3	**11**	**10**
	15	4-4	1-1	1-1	0-0	0-0	0-0	7-7	13	13
DVT	16	4-4	1-1	0-0	0-0	0-0	0-0	7-7	12	12
	17	4-4	0-0	0-0	0-0	0-0	0-0	7-7	11	11
	18	4-4	2-2	1-1	0-0	0-0	0-0	0-0	7	7
	19	4-4	3-3	2-2	0-0	0-0	0-0	7-7	16	16
	20	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5
	21	4-4	1-1	1-1	0-0	0-0	0-0	3-3	9	9
	22	4-4	2-2	4-4	0-0	0-0	0-0	3-3	13	13
DVT	23	4-4	2-2	0-0	0-0	0-0	0-0	0-0	6	6
DVT	24	4-4	2-2	2-2	0-0	0-0	0-0	3-3	11	11
	25	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5

Percentage agreement:

$$\frac{23}{25}\times100=92\%$$

Table 5.8: Agreement matrix on medical ward 30

A \ B	No risk	Low risk	Moderate risk	High risk	Total
No risk	8	1	-	-	9
Low risk	-	7	-	-	7
Moderate risk	-	1	7	-	8
High risk	-	-	-	1	1
Total	8	9	7	1	25

Observed agreement: $\frac{23}{25} = 0.92$

Expected agreement $\frac{8\times9}{25} + \frac{9\times7}{25} + \frac{7\times8}{25} + \frac{1\times1}{25}$

$$= \frac{2.88}{25} + \frac{2.52}{25} + \frac{2.24}{25} + \frac{0.04}{25} = \frac{7.68}{25} = 0.31$$

$$K = \frac{0.92 - 0.31}{1 - 0.31} = \frac{0.61}{0.69} = 0.88$$

kappa value on ward 30 = 0.88.

Table 5.9: Reliability study on ward 27 (Surgical directorate)

Assessor A-B	Patients	Age	Build	Mobility	Special risk	Trauma	Surgery	High Risk Diseases	Assesor A	Assesor B
									Total Scores	
DVT	1	4-4	2-2	0-0	0-0	0-0	1-1	7-7	14	14
DVT	2	2-2	2-2	2-2	0-0	0-0	2-2	0-0	8	8
	3	0-0	1-1	0-0	0-0	0-0	3-3	0-0	4	4
	4	1-1	1-1	0-0	0-0	0-0	3-3	0-0	5	5
DVT	5	3-3	2-2	0-0	0-0	0-0	3-3	0-0	8	8
	6	3-3	2-2	0-0	0-0	0-0	1-1	0-0	6	6
	7	2-2	2-2	0-0	0-0	0-0	1-1	3-3	8	8
	8	1-1	2-2	0-0	0-0	0-0	1-1	0-0	4	4
	9	4-4	2-2	1-1	0-0	0-0	2-2	0-0	9	9
	10	3-3	2-1	2-2	0-0	0-0	3-3	1-1	11	10
	11	0-0	0-0	0-0	0-0	0-0	1-1	0-0	1	1
	12	4-4	1-1	1-1	0-0	0-0	3-3	0-0	9	9
	13	4-4	1-1	2-2	0-0	0-0	3-3	0-0	10	10
	14	2-2	1-1	1-1	0-0	0-0	3-3	0-0	7	7
	15	4-4	1-1	2-2	0-0	0-0	3-3	0-0	10	10
DVT	16	4-4	1-1	2-2	0-0	0-0	3-3	5-5	15	15
	17	4-4	1-1	1-1	0-0	0-0	3-3	0-0	9	9
	18	4-4	2-2	0-0	0-0	0-0	3-3	4-4	13	13
	19	4-4	1-1	2-2	0-0	0-0	0-0	3-3	10	10
	20	0-0	3-3	1-1	0-0	0-0	2-2	0-0	6	6
	21	4-4	1-1	2-2	0-0	0-0	3-3	0-0	10	10
	22	4-4	1-1	0-0	0-0	0-0	0-0	6-6	11	11
	23	1-1	3-3	0-0	0-0	0-0	2-2	0-0	6	6
	24	4-4	1-1	0-0	0-0	0-0	0-0	3-3	8	8
DVT	25	4-4	1-1	2-2	0-0	0-0	0-0	10-10	17	17
	26	4-4	0-0	2-2	0-0	0-0	0-0	0-0	6	6
	27	4-4	0-0	0-0	0-0	0-0	0-0	7-7	11	11
	28	1-1	1-1	0-0	0-0	0-0	3-3	0-0	5	5
	29	4-4	1-1	2-2	0-0	0-0	3-3	0-0	10	10
	30	4-4	2-2	3-3	0-0	0-0	3-3	0-0	12	12
	31	1-1	0-0	4-4	0-0	0-0	0-0	0-0	5	5
	32	4-4	3-3	1-1	0-0	0-0	1-1	0-0	9	9
DVT	33	4-4	1-1	0-0	0-0	0-0	3-3	3-3	11	11
	34	4-4	2-2	4-4	0-0	0-0	0-0	7-7	17	17
	35	3-3	1-1	0-0	0-0	0-0	3-3	0-0	7	7
	36	4-4	1-1	0-0	0-0	0-0	3-3	0-0	8	8
	37	4-4	0-0	1-1	0-0	0-0	0-0	5-5	10	10
	38	4-4	0-0	0-0	0-0	0-0	0-0	5-5	9	9
DVT	39	4-4	3-3	0-0	0-0	0-0	0-0	5-5	12	12
	40	1-1	1-1	0-0	0-0	0-0	0-0	7-7	9	9
	41	4-4	4-4	0-0	0-0	0-0	0-0	0-0	8	8
	42	2-2	1-1	2-2	0-0	0-0	3-3	0-0	8	8
	43	4-4	1-1	2-2	0-0	0-0	3-3	0-0	10	10

Assessor A-B	Patients	Age	Build	Mobility	Special risk	Trauma	Surgery	High Risk Diseases	Assesor A	Assesor B
									Total Scores	
	44	4-4	0-0	2-2	0-0	0-0	3-3	0-0	10	10
	45	3-3	1-1	0-0	0-0	0-0	0-0	0-0	4	4
DVT	46	4-4	1-1	0-0	0-0	0-0	2-2	6-6	13	13
	47	3-3	2-2	0-0	0-0	0-0	1-1	3-3	9	9
	48	4-4	2-2	0-0	0-0	0-0	1-1	0-0	7	7
	49	3-3	2-2	0-0	0-0	0-0	3-3	0-0	8	8
	50	4-4	1-1	0-0	0-0	0-0	0-0	0-0	5	5

Percentage agreement:

$$\frac{49}{50}\times 100 = 98\%$$

Table 5.10: Agreement matrix on the surgical ward.

B / A	No risk	Low risk	Moderate risk	High risk	Total
No risk	12	-	-	-	12
Low risk	-	26	1	-	27
Moderate risk	-	-	8	-	8
High risk	-	-	-	3	3
Total	12	26	9	3	50

Observed agreement $(\text{Po}) = \frac{49}{50} = 98$

Expected agreement:

$$(\text{Pe}) = \frac{12\times 12}{50} + \frac{26\times 27}{50} + \frac{9\times 8}{50} + \frac{3\times 3}{50} = \frac{2.9+14.04+1.4+0.18}{50} = 0.37$$

$$K = \frac{0.96-0.37}{1-0.37} = \frac{0.59}{0.63} = 0.94$$

kappa value on ward 27 = 0.94

Summary & interpretation of results

There is a consensus that a minimum of 70 per cent agreement is necessary, 80 per cent is adequate and 90 per cent is good (Hartmann et al, House et al, 1981). Total percentage agreements within the five reliability studies were high and ranged from 85- 98 per cent.

Like a correlation coefficient, kappa values vary from –1.0 for complete disagreement, 0 for chance agreement to +1 for perfect agreement. The kappa values for the same five reliability studies ranged from 0.88 - 0.95, confirming an almost perfect agreement as tabulated below by the heuristic for the various values of k (Landis and Koch, 1977). Expected values for chance correction or adjustment ranged from 0.27-0.38, thus further substantiating the high kappa values reported.

Table 5.11: Interpretation of various values of kappa

Kappa statistic	Strength of agreement
< 0.00	Poor agreement
0.00-0.20	Slight agreement
0.21-40	Fair agreement
0.41-0.60	Moderate agreement
0.61-0.80	Substantial agreement
0.81-1.00	Almost perfect agreement

Landis and Koch (1977) admit that the boundaries between the adjacent categories are arbitrary, albeit useful benchmarks. What constitutes a good kappa value is controversial. Landis and Koch (1977) claim a substantial agreement for a kappa value 0.61 but other researchers have insisted on a kappa value of no lower than 0.75 (Mehrens & Lehmans, 1978; Blenner, 1993).

Intra-class correlation coefficient (ICC)

Further to substantiate the high inter rater reliability of the DVT scale, ICC was computed for all the five clinical areas. ICC estimates the average correlation among all possible pairs of observation (Bland & Altman, 1996; Anthony, 1999). It takes into account the error between two raters in both their absolute scores and the ranking of scores (Tomalin et al, 1993).

As the number of observation was the same for each subject recorded by paired assessors, ICC was considered appropriate. Bland & Altman (1996) suggest that the following equation be applied to calculate ICC, when a computer software package is unavailable:

$$r_1 = \frac{mSS_b - SS_t}{(m-1)\,SSt}$$

Where: r1 is the intra-class correlation coefficient

m is the number of observation

SS_b is the between subjects sum of squares

SS_t is the total sum of squares

The available data from Tables 5.1, 5.3, 5.5. 5.7 and 5.9 used to estimate the total percentage agreement and kappa values, were computed to estimate ICC, using SPSS (version 11) via statistic pull down and selection of intra-class correlation coefficient (Table 5.12).

Table 5.12: Intra-class correlation coefficients analysis

Intra-class correlation coefficients:Ward C

```
 Method 1 (space saver) will be used for this analysis.

 R E L I A B I L I T Y   A N A L Y S I S   - S C A L E (A L P H A)

Intraclass Correlation Coefficient
One-way random effect model: People Effect Random
 Single Measure Intraclass Correlation =     .9975
    95.00% C.I.:            Lower =    .9945          Upper =
.9989
 F = 803.8800   DF = (    25,   26.0)   Sig. = .0000  (Test Value =
.0000 )
 Average Measure Intraclass Correlation =     .9988
    95.00% C.I.:            Lower =    .9973          Upper =
.9994
 F = 803.8800   DF = (    25,   26.0)   Sig. = .0000  (Test Value =
.0000 )

Reliability Coefficients

N of Cases =     26.0                    N of Items =  2

Alpha =    .9987
```

Intra-class correlation coefficients: Ward D

```
* Method 1 (space saver) will be used for this analysis.
 R E L I A B I L I T Y   A N A L Y S I S  - S C A L E   (A L P H A)

Intraclass Correlation Coefficient
One-way random effect model: People Effect Random
 Single Measure Intraclass Correlation =     .8967
    95.00% C.I.:            Lower =     .7789          Upper =
.9537
 F =  18.3564   DF = (     23,    24.0)   Sig. = .0000  (Test Value =
.0000 )
 Average Measure Intraclass Correlation =     .9455
    95.00% C.I.:            Lower =     .8757          Upper =
.9763
 F =  18.3564   DF = (     23,    24.0)   Sig. = .0000  (Test Value =
.0000 )

Reliability Coefficients

N of Cases =     24.0                    N of Items =  2

Alpha =     .9445
```

Intra-class correlation coefficients: Ward 29

```
* Method 1 (space saver) will be used for this analysis.

 R E L I A B I L I T Y   A N A L Y S I S  - S C A L E   (A L P H A)

Intraclass Correlation Coefficient
One-way random effect model: People Effect Random
 Single Measure Intraclass Correlation =     .9976
    95.00% C.I.:            Lower =     .9947          Upper =
.9989
 F = 836.1250   DF = (     24,    25.0)   Sig. = .0000  (Test Value =
.0000 )
 Average Measure Intraclass Correlation =     .9988
    95.00% C.I.:            Lower =     .9973          Upper =
.9995
 F = 836.1250   DF = (     24,    25.0)   Sig. = .0000  (Test Value =
.0000 )

Reliability Coefficients

N of Cases =     25.0                    N of Items =  2

Alpha =     .9988
```

Intra-class correlation coefficients: Ward 30

```
* Method 1 (space saver) will be used for this analysis.
 R E L I A B I L I T Y   A N A L Y S I S - S C A L E   (A L P H A)

Intraclass Correlation Coefficient
One-way random effect model: People Effect Random
 Single Measure Intraclass Correlation =     .9966
    95.00% C.I.:            Lower =    .9923          Upper =
.9985
 F = 580.9583   DF = (    24,   25.0)   Sig. = .0000  (Test Value =
.0000 )
 Average Measure Intraclass Correlation =     .9983
    95.00% C.I.:            Lower =    .9961          Upper =
.9992
 F = 580.9583   DF = (    24,   25.0)   Sig. = .0000  (Test Value =
.0000 )

Reliability Coefficients

N of Cases =     25.0                    N of Items =  2

Alpha =    .9984
```

Intra-class correlation coefficients: ward 27

```
* Method 1 (space saver) will be used for this analysis.
R E L I A B I L I T Y   A N A L Y S I S  -  S C A L E   (A L P H A)

Intraclass Correlation Coefficient
One-way random effect model: People Effect Random
 Single Measure Intraclass Correlation =     .9991
    95.00% C.I.:            Lower =    .9983          Upper =
.9995
 F = 2117.571   DF = (    49,   50.0)   Sig. = .0000  (Test Value =
.0000 )
 Average Measure Intraclass Correlation =     .9995
    95.00% C.I.:            Lower =    .9992          Upper =
.9997
 F = 2117.571   DF = (    49,   50.0)   Sig. = .0000  (Test Value =
.0000 )

Reliability Coefficients

N of Cases =     50.0                    N of Items =  2

Alpha =    .9995
```

The ICCs and their significance level for each ward are summarised below:

Table 5.13: Summary of Intra-class correlation coefficients on all wards.

Clinical areas	ICC value (Alpha)	Significance level
Ward c (orthopaedics)	.9987	< 0.0001
Ward d (orthopaedics)	.9445	< 0.0001
Ward 29 (medicine)	.9988	< 0.0001
Ward 30 (medicine)	.9984	< 0.0001
Ward 27 (surgery)	.9995	< 0.0001

Interpretation of the reliability coefficient

There is no standard for what an acceptable reliability coefficient should be (Polit & Hungler, 1999). A coefficient in the vicinity of .75 specified by Ventura et al (1980) might be sufficient, although coefficient of .80 or greater is highly desirable.

On the other hand, if measures are to be used as a basis for making decision about an individual, then the reliability coefficient ideally should be .90 or better. The high ICCs recorded in all five clinical areas mirror the high percentage agreement and kappa values and are well above the criterion of .75 specified by Ventura et al, 1980.

Sensitivity and specificity of the Autar DVT scale

Epidemiological methods are available to predict that a tool is truly able to discriminate between individuals with and without a particular characteristic. The prime factor to consider in the selection of a predictive index or a diagnostic tool is its accuracy. Wheeler et al (1994) identify four components of test accuracy which are:

- Sensitivity
- Specificity
- Positive Predictive Value (PPV)
- Negative Predictive Value (NPV)

All of the four measurements were applied to evaluate the clinimetric properties and predictive accuracy of the DVT scale.

Sensitivity relates to the proportion of patients with the disease (DVT) and who were correctly predicted to be at risk.

Sensitivity Formula:

$$= \frac{\text{True Positive}}{\text{True Positive} + \text{False Negative}} \times 100$$

(All individuals with the disease)

Specificity is the proportion of patients without the condition (DVT) and who were predicted not to be at risk.

Table 5.14: Diagnostic clinical model for pretest probability of DVT (Wells et al 1995)

Clinical manifestation	Score
Active cancer(treatment ongoing or within 6 months or palliative)	1
Paralysis, paresis or recent immobilisation of the legs	1
Recent immobilisation >3 days or major surgery within 12 weeks requiring general or regional anaesthesia	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling > 3 cm than asymptomatic side (measured 10 cm below tibial tuberosity)	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Alternative diagnosis equally or more likely than DVT	-2

Assessment Protocol:
High risk= 3 points Moderate risk=2 points Low = 0 point

In approximately 70 per cent of patients with clinically suspected DVT, alternative diagnoses are ultimately found as follows:

- Achilles tendonitis
- Arterial insufficiency
- Rheumatoid arthritis
- Peripheral oedema due to chronic heart disease, liver or renal failure
- Cellulitis
- Lymphangitis
- Haematoma
- Lymphodema
- Muscle or soft tissue injury
- Post phlebitis syndrome
- Limb paralysis
- Ruptured Baker cyst
- Superficial phlebitis
- Varicose veins

Data: emedicine. com.inc, 2001.

This pretest probability model enables clinicians to reliably stratify their patients into high, moderate and low risk categories. Combining this with the

Specificity Formula:

$$= \frac{\text{True Negative}}{\text{True Negative} + \text{False Positive}} \times 100$$

(All patients without the disease)

Sensitivity and specificity are useful and practical statistics in as much as they yield consistency of result. Sensitivity is conditional on the disease being present and specificity on being absent. Both sensitivity and specificity are useful tests because they are unaffected by disease prevalence. If the number of subjects is doubled, then twice as many patients will give a particular outcome. Sensitivity and specificity would be unchanged from previous values and therefore yield a consistent result for the diagnostic test in a variety of patient group with different disease prevalence (Campbell & Machin,1993). In brief, sensitivity and specificity are characteristics of the test and not the population to which it is applied.

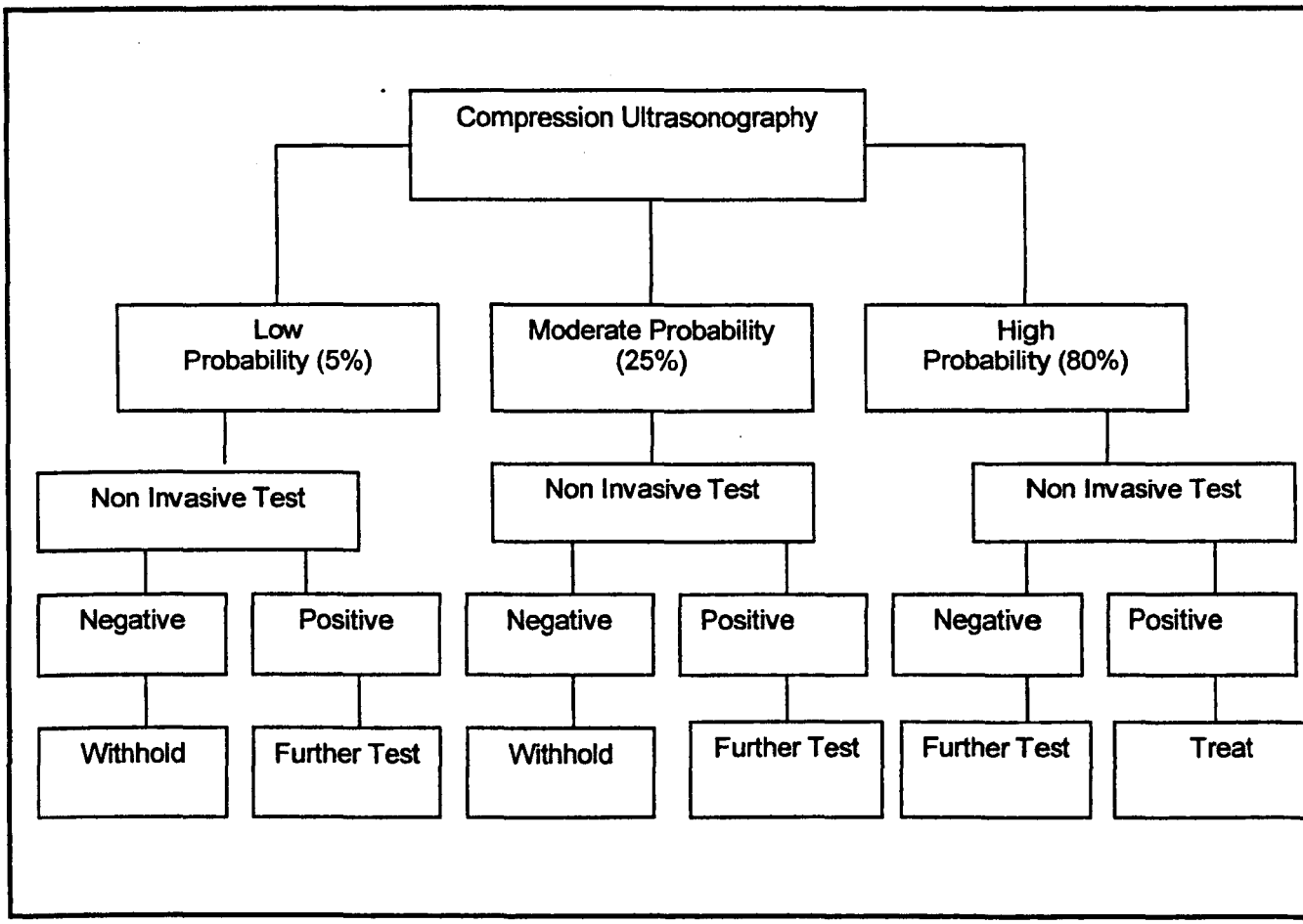
To estimate the sensitivity and specificity of the Autar DVT scale, data gathered prospectively from the 150 patients from the orthopaedic, medical and surgical directorates were pooled for analysis .Two patients were excluded from the sensitivity and specificity data analysis, as it was not possible to follow them up from hospital on discharge to no fixed abodes. Irrespective of primary prophylaxis, all subjects were closely monitored for occurrence of DVT. Patients who were suspected of DVT were duly investigated by following the established trust screening protocol, before secondary venous thromboprophylaxis was administered for DVT treatment.

The diagnostic clinical predictive guide developed by Wells et al (1994) was adopted by the trust as a protocol and applied to all patients suspected of DVT. This diagnostic tool quantifies the pretest probability of DVT (Table 5.14) The Wells et al clinical prediction guide incorporates risk factors, clinical signs and the presence and absence of alternative diagnoses.

results of objective testing provides an algorithm that greatly simplifies the clinical work-up of patients suspected of DVT (Table 5.15).

Table 5.15: An algorithm for the investigation of suspected DVT

Diagnostic Clinical Probability of suspected DVT



Wells et al (1993 & 1994) evaluated the clinical model in patients suspected of DVT in order to determine their pretest probability of the disease. The pretest clinical probability was classified as low, moderate and high based on a simple scoring system (Table 5.12). After determining the pretest probability, the 243 patients had a venogram and the findings were interpreted without the knowledge of the clinical groupings. An overall prevalence of 20 per cent (49) DVT was recorded. Patients classified as having low probability of DVT constituted 60 per cent (145) of the total population and the prevalence of DVT in this large group was only 5 per cent. In patients classified as having moderate pretest probability of DVT, a prevalence of nearly 25 per cent was recorded. In the high pretest clinical

probability group which constituted only 15% of the total population, the prevalence was nearly 80 per cent (28/36)

A clinical prediction rule must clearly define the event to be predicted and such definition must be free from bias and ambiguity (Wasson et al, 1985). In this prospective study, the predictive rule was that the patients predicted to develop DVT ultimately contracted this silent and asymptomatic condition. For the purpose of statistical analysis of the DVT scale for its sensitivity and specificity, the gold standard was defined as patients with a confirmed diagnosis of DVT treated with secondary anticoagulant therapy. Twenty-eight such subjects met the gold standard. All the patients were closely monitored for occurrence of DVT on the ward as well as followed up for three months after discharge home. DVT is a continuing problem as risk factors persist well after discharge from hospital (Scurr et al,1988; Scurr, 1990; Anderson et al, 1995). Some patients become rather complacent and less mobile and 25 percent of patients (13/51) develop DVT within six weeks of discharge from hospital (Scurr et al, 1988). This report is further substantiated by the clinical findings that 11 out of the 28 patients (39 %) developed DVT at home (Tables 5.31, 5.32 & 5.34).

The DVT risk assessment scores of all patients from the orthopaedics, medical and surgical directorates were computed to establish the sensitivity and specificity of the DVT scale.

Overall, the convenience sample yielded a DVT prevalence of 19% (28/148) and is tabulated below:

Table 5.16: Incidence of DVT on the three clinical specialities

Speciality	No of patients	No of DVT	%
Orthopaedic Trauma	50	8	16
Medical	50	12	24
Surgical	48	8	17
Total	148	28	19

The score values of all patients with and without DVT is exhibited in table 5.17.

Table 5.17: Score values of patients with and without DVT. Raw data converted to percentages.

%	DVT present	Scores	DVT absent	%
0	0	1	2	2
0	0	2	2	2
0	0	4	8	7
7	2	5	14	12
7	2	6	12	10
3.5	1	7	6	5
11	3	8	11	9
3.5	1	9	13	11
0	0	10	15	13
11	3	11	11	9
11	3	12	5	4
14	4	13	8	7
7	2	14	1	1
7	2	15	3	2.5
7	2	16	3	2.5
3.5	1	17	2	2
0	0	19	1	1
3.5	1	20	2	2
3.5	1	26	0	0
0	0	27	1	1
100%	28		120	100%

N = 148

Most clinical data have a fair degree of overlap (Sackett et al, 1991). A tied score is of interest when a diseased subject has the same test result as a non diseased individual (Zweig & Campbell, 1993). Interestingly, within the medical directorate, two patients with a tied score of 5 had DVT and fourteen did not. Close scrutiny of the sensitivity and specificity data reveals that although several patients in the orthopaedic and surgical directorates also had tied scores of 5, none of them as correctly predicted developed DVT. Patients who developed DVT in the latter directorates had scores ranging within the bound of moderate to high-risk categories.

On the medical directorate within the tied score of 5 between subjects with and without DVT, the event was seemingly coincidental, albeit advancing age appeared to be a powerful discriminating risk factor. It is possible that the nature and extent of nursing intervention given to those patients with a tied score of 5 was influential of the outcome. Some subjects might have

attracted more attention than others, resulting in receiving little or more nursing intervention, directed at preventing DVT.

Equally, some patients with recorded tied scores of 15≥ developed DVT and others did not. The tied scores of 15≥ with and without DVT across the clinical specialities are exhibited in table 5.18.

Table 5.18: Tied scores of 15≥

Speciality	DVT present	DVT absent
Orthopaedics	4	7
Medical	1	3
General Surgery	2	2

A likely explanation is that the patients with the tied score of 15 ≥ did not develop DVT, irrespective of pharmacological prophylaxis, on account of aggressive DVT nursing interventions.

Nurses are at the forefront of assessment, planning, care delivery and the evaluation processes. By virtue of having identified patients at high risk of DVT, it followed that they would "act always in such a manner as to promote and safeguard the interest and well being of patients or clients" and therefore:
"ensure that no action or omission on your part, or within your sphere of responsibility, is detrimental to the interests, condition or safety of patients and clients" (UKCC, 1992).
DVT nursing interventions in the form of mechanical prophylaxis such as active physiotherapy, ambulation, leg elevation and application of graduated elasticated compression stockings are effective measures at preventing DVT even in high risk group, with no adverse effect.

On the medical directorate coexisting high risk diseases such as myocardial infarction and chronic heart diseases were associated with the occurrence of DVT.

In the trauma / orthopaedic directorate, advanced age, immobility due to fracture neck of femur and the nature of surgical procedures to repair the

damage were significant risk variables in the causation of DVT in some patients.

On the surgical directorate, elderly patients who underwent abdominal surgery for malignancies, were the highest risk group. One patient with inoperable malignancy of the bowel and receiving palliative care developed DVT.

All patients across the clinical specialities with previous DVT had recurrence of the condition, thus confirming outcome of previous studies that recurrence rate is as high as 68 per cent (Nicolaides & Irving, 1975; Wheeler, 1988).

High-risk patients carry an absolute risk of DVT ranging between 40-80% (Weinmann and Saltzmann, 1994). The absolute score of ≥ 15 of the assessment protocol was chosen to represent the high risk group and calculate the sensitivity and specificity of the DVT scale. The sensitivity and specificity of the absolute cutoff value is illustrated in the contingency table below:

Table 5.19: The Sensitivity and specificity of absolute scores in DVT.

	DVT present	DVT absent	Total
Score 15 ≥	A 7	B 12	19
Score ≤ 14	C 21	D 108	129
Total	28	120	148

$$\text{Sensitivity: } \frac{a}{a+c} \times 100 = \frac{7}{7+21} \times 100 = 25\%$$

$$\text{Specificity: } \frac{d}{b+d} \times 100 = \frac{108}{12+108} \times 100 = 90\%$$

A highly sensitive tool will rarely miss people with disease and conversely a specific instrument will rarely misclassify people without the disease as diseased (Fletcher, Fletcher & Wagner, 1988).
True positives are those who are predicted as positive and have the disease. Seven patients were correctly classified as true positive.

False positives are those who are incorrectly predicted to have the disease when they do not. Twelve patients belonged to the score range of ≥ 15 but did not develop DVT as predicted.

Those who are predicted negative and who do not have the disease are the true negatives and 108 patients were correctly classified.

False negatives are those subjects who are predicted negative for the disease but contracted it. 21 false negatives were recorded.

The DVT scale achieved a sensitivity of only 25 % in contrast to the remarkably high specificity of 90% of patients who did not develop DVT and were correctly predicted.

Sensitivity and specificity are inversely related (Mausner & Kramer 1985). A high specificity of 90% was achieved at the expense of a low sensitivity of only 25%.

Test accuracy is defined as the number of true positive and true negative divided by the number of patients studied (Wheeler et al, 1994).Overall, using the absolute score range, the DVT scale correctly classified 115 patients
(7 TP+ 108TN/148=78%) and misclassified 33 patients (22%). 14% of the misclassified patients developed DVT.

Apparently, there are two problems arising from the application of the absolute score. First of all, concomitantly, the decreed score of ≥ 15 ruled in only 25% of patients with DVT and ruled out 75% with the predicted target disorder (Table 5.17).

Secondly, there is no guarantee that the absolute score would generate similar distribution of scores in the cohort of the next 150 patients. Invariably, as more and more patients are sampled, the range of scores can only get bigger and probably more patients without DVT will exhibit higher and higher scores. Consequently, one would be forced to move the absolute score to an even greater extreme level to predict patient with DVT.

On the other hand, if the score were lowered, say ≥ 11, more patients would reside within this DVT risk category and would encompass 68% compared to the previously recorded 25% (Table 5.17).

On account of the limitations presented by the absolute score value, in predicting patients for DVT risk, the four cutoff values for the four risk categories in the Autar DVT scale assessment protocol were also calculated. Table 5.20 highlights the distribution of patients with and without DVT by cutoff score values and table 5.21 exhibits the sensitivity and specificity for the four cutoff values, as illustrated in table 5.17.

Table 5.20: Distribution of non DVT and DVT patients by cutoff score values

Cutoff values	DVT absent	DVT present
≥15 High Risk	12	7
11-14 Moderate risk	25	12
7-10 Low risk	45	5
≤6 No risk	38	4
Total	120	28

Table 5.21: Sensitivity and specificity for the four cutoff values

Cutoff scores	High risk ≥15	Moderate risk 11-14	Low risk 7-10	No risk ≤6
Sensitivity	7/28= 25%	(12+7)/28=68%	(5+12+7)/28=86%	(4+5+12+7)/28=100%
Specificity	12/120=10%	(12+25)/120=31%	(45+25+12)/120= 68%	(120/120)=100%
1-Specificity	.90	.69	.32	.0

The choice of 11-14 score range optimises the predictive potential of the DVT scale and allocate subjects to a specific risk category and deals with the problem of outliers and subjects belonging to" no man's land" (Sackett et al 1991). A sensitivity of 68 per cent was recorded.

Overall, fewer patients in the high risk score range were recruited. The distribution of patients in each of the four score ranges is illustrated in table 5.22.

Table 5.22: Distribution of patients by risk categories

Score range	No of patients	%
≥ 15	19	13
11-14	37	25
7-10	50	34
≤ 6	42	28

Although sensitivity and specificity provide consistency and information about the accuracy of the test, they do not add to the meaning of positive and negative test results, While sensitivity and specificity are conditioned by the knowledge of the disease state, predictive values are conditioned by the nature of the test result (Essex-Sorlie, 1995).

Predictive values were therefore used to measure the frequency with which the DVT scale correctly identified those at risk.
Positive Predictive Values (PPV) are the proportion of testing positive or predicted at risk of DVT, who actually develop DVT. This is summarised by:

$$\frac{\text{True Positive}}{\text{True Positive} + \text{False Positive}} \times 100$$

Negative Predictive Values NPV) is the proportion of those predicted negative who do not have DVT. This is expressed as:

$$\frac{\text{True Negative}}{\text{True Negative} + \text{False Negative}} \times 100$$

PPV and NPV for the absolute score is shown in table 5.23

Table 5.23: The Predictive values of the DVT scale for the absolute score range

	DVT present	DVT absent	Total	Predictive values
Score values ≥ 15	True positive 7	False Positive 12	19	Positive Predictive Value: 37%
≤ 14	False negative 21	True negative 108	129	Negative predictive Value: 84%
Total	28	120	148	

Difference between prior probability (prevalence) and posterior probability (predictive value) is one way of assessing the usefulness of a test

(Campbell & Machin, 1994). A DVT prevalence of 19% was recorded and predictive +ve value almost doubled (37%).

However, considerable caution must be exercised in the interpretation of predictive values, as there are two factors that influence the predictive values of an instrument: prevalence of the target disorder and sensitivity and specificity of the tool (Essex-Sorlie, 1994).

"As prevalence falls, positive predictive value must fall alongside it, and negative predictive value must rise"
(Sackett et al, 1991:p 88.

If the prevalence of the disease is low, the positive predictive value will not be close to 1 even if the sensitivity and specificity are high. It is therefore inevitable that many people predicted positive will be false positive (Altman & Bland, 1994).
A fall in PPV (37%) due to low prevalence of DVT (19%) was associated with a concomitant rise in NPV (84%).

Also, predictive values observed in one study do not apply universally (Altman & Bland, 1994). An instrument with a high predictive value in one clinical setting may be of little predictive utility in another area, because of the difference in prevalence in the two populations. For instance, the prevalence of DVT in the medical patients ranges between 13-16 % (Kierkegaard, 1987) and approximately 25% in the surgical population (Clagett & Reisch, 1988) compared to 40-80 in the orthopaedic population (Weinmann & Salzman, 1994).
When prevalence is low, the greater are the false positives. 12 subjects were classified as false positive.

Conversely, when a highly sensitive test is applied to a population where the prevalence is high, negative test results will be largely false negatives. Twenty-one false negatives were identified.

There is also a relationship between the sensitivity and specificity and the predictive value of an index. When the sensitivity of an instrument increases, it is less likely that an individual with a negative prediction will have DVT. Thus, the greater the sensitivity of a test, the higher is the

negative predictive value. On the other hand, if the specificity of the test increases, its positive predictive value is increased. Diagnostic tools require the highest levels of sensitivity and specificity (Larsen 1986). But the same level is not expected of a screening tool and is even less likely to occur when the condition is preventable (Morris 1985). Even if the DVT scale accurately predicts those at risk, patients who receive care that successfully prevents DVT become classified as false positives. Screening tests are therefore not designed to be diagnostic but to identify patients with a high probability of having the disease (Larsen, 1986).

Norton (1989) is satisfied that measures of predictive validity should not be rigidly applied to risk assessment scales. She argues that it is a more reasonable approach to hold a risk assessment scale to a level of predictive efficiency for any screening test rather than that of a diagnostic tool. Some instruments may fail to address some issues but this does not necessarily invalidate the tool because of unrealistic expectation of what can be achieved with a single instrument (Gibbon, 1998). Perhaps a risk assessment tool should carry a health warning that it should be used in conjunction with clinical judgement.

Cutoff scores

A sensitive tool is one that correctly identifies or predicts those with the condition as well as those without. However, 100 per cent test accuracy or prediction is unachievable and invariably there will be some overlap and misclassification of the predictive outcomes (Larsen, 1986). Some high risk patients who are predicted to develop DVT may not and others who are not may do so. A sensitive measure at the expense of specificity is not necessarily a better one (Anthony, 1999).

A perfect prediction would be no false positive or false negative outcomes. Since sensitivity and specificity are inversely correlated (Lilienfield & Lilienfield 1980; Mausner & Kramer, 1985) increasing sensitivity by lowering the score at which a prediction is considered possible, decreases the specificity of the instrument and vice versa.

The complex tradeoffs between sensitivity require consideration of the following values:

- True positive
- True negative
- False positive
- False negative

Cost of true positive: The whole point of a diagnostic tool is to correctly make prediction about the target disorder (Altman, 1994). Predicting a true positive means that the patients who are potential subject for DVT will be prescribed primary prophylaxis in the anticipation that this will prevent DVT or be seen to minimise the risk.

Cost of true negative: The benefit of true negative implies that patients who are not at risk of DVT will not unnecessarily commence primary pharmacological prophylaxis and be at risk of iatrogenic bleeding. Limited resources can then be allocated to those who need them most.

Cost of false positive: A false positive prediction can place the patients at risk by unnecessarily being administered pharmacological prophylaxis. They may be at risk of iatrogenic bleeding. However, choosing the more benign mechanical venous thromboprophylaxis over a pharmacological one eliminates this problem.

Cost of false negative: The subjects are initially predicted not to be at risk and therefore do not require prophylaxis against DVT. The consequential penalty of this misclassification is the potential development of DVT, which is preventable by the appropriate venous thromboprophylaxis. Additionally, a false negative could carry the risk of litigation due to clinical omission and negligence (Parker-Williams & Vickers, 1989). Such cases are on the increase (NAHAT, 1991).

The sensitivity and specificity of the DVT scale is dependent on the threshold used. However, tests do not have only one sensitivity and specificity. Calculating just one pair of sensitivity and specificity provides only a very brief glimpse of a test performance.The sensitivity and specificity of the absolute score for the DVT scale only achieved 25% sensitivity but 90

% specificity (Table 5.19), using a ≥ 15 cutoff value. This might be far from revealing of the real diagnostic abilities of the DVT risk calculator.

The application of an absolute cutoff score range of ≥ 15 ruled in only 25 per cent of patients with DVT and ruled out 75 per cent of the cases (Table 5.17). On the other hand, lowering the score range to ≥ 11 captures 68 per cent of patients with DVT. Data from tables 5.1,5.3,5.5,5.7 and 5.9 representing the five wards of the trust were analysed to compare the outcome of a cutoff score of ≥ 11 with the cutoff score range of ≥ 15. On all five wards, the cutoff score of ≥ 11 performs better in terms of negative predictive validity of the DVT scale (Tables 5.24 –5.28).

Table 5.24:Cutoff scores on orthopaedic ward C. No of patients: 26. Total DVT recorded: 3

DVT cutoff	TP	FP	TN	FN	PPV	NPV
11 ≥	3	9	14	0	25%	100%
15 ≥	3	1	22	0	75%	100%

Table 5.25:Cutoff scores on orthopaedic ward D. No of Patients 24. Total DVT recorded: 5

DVT cutoff	TP	FP	TN	FN	PPV	NPV
11≥	4	13	6	1	24%	86%
15 ≥	1	6	14	4	14%	50%

Table 5.26:Cutoff scores on medical directorate ward 29. No of patients: 25. Total DVT recorded: 6

DVT cutoff	TP	FP	TN	FN	PPV	NPV
11 ≥	3	3	16	3	50%	84%
15 ≥	1	2	17	5	33%	77%

Table 5.27:Cutoff scores on medical directorate ward 30. No of Patients: 25. Total DVT recorded: 6

DVT cutoff	TP	FP	TN	FN	PPV	NPV
11 ≥	3	6	13	3	33%	81%
15 ≥	0	1	18	6	---	75%

Table 5. 28 :Cutoff scores on surgical ward 27: No of patients: 48 out of 50. Total DVT recorded: 8

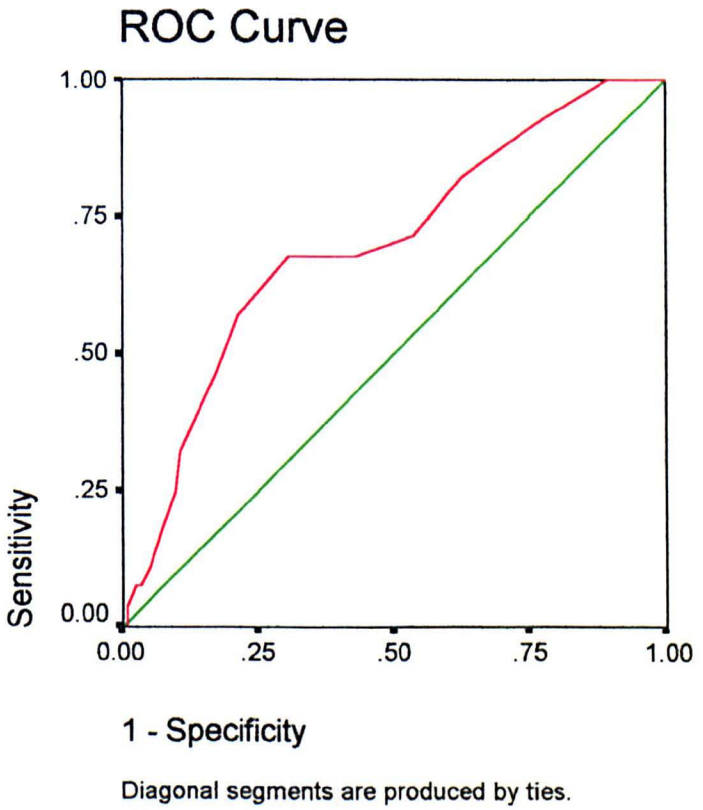
DVT cutoff	TP	FP	TN	FN	PPV	NPV
11≥	6	5	36	1	55%	97%
15 ≥	2	1	40	5	66%	89%

Although a decision threshold must be chosen for risk assessment and management, it is undesirable to choose any particular decision threshold for assessing accuracy. Assessing a single point may result in misleading impression about the test performance (Zweig & Campbell, 1993).

Receiver Operating Characteristics (ROC)

In order to address the limitation of selecting a single pair of sensitivity and specificity values, several thresholds were applied in the construction of a Receiver Operating Characteristics (ROC) curve for optimal predictive accuracy of the DVT scale. A ROC (figure 6) is simply a plot of the true positive rate against the false positive rate for the given thresholds and "is a fundamental evaluation tool in clinical medicine" (Zweig & Campbell, 1993). It is based on the statistical decision theory, developed in the 1950's for radar signal detection. It is a method of assessing the sensitivity and specificity of a classification at a variety of thresholds, allowing for a qualitative comparison of several classifiers (Anthony, 1999).

Figure 6: Receiver Operating Characteristics (ROC)



Area Under the Curve

Test Result Variable(s): SCORE

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.696	.056	.001	.587	.806

The test result variable(s): SCORE has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Coordinates of the Curve

Test Result Variable(s): SCORE

Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity
.00	1.000	1.000
1.50	1.000	.983
3.00	1.000	.967
4.50	1.000	.893
5.50	.929	.777
6.50	.857	.678
7.50	.821	.628
8.50	.714	.537
9.50	.679	.430
10.50	.679	.306
11.50	.571	.215
12.50	.464	.174
13.50	.321	.107
14.50	.250	.099
15.50	.179	.074
16.50	.107	.050
18.00	.071	.033
19.50	.071	.025
23.00	.036	.008
26.50	.000	.008
28.00	.000	.000

The test result variable(s): SCORE has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

Using the version 11 of the SSPS (2001) data on 148 patients were analysed to construct the ROC, DVT being the state variable. ROC analysis provides a graphic view of the whole spectrum of sensitivity and specificity decision at the various selected thresholds.

The ROC curve that is closest to the upper left hand corner is the best cutoff score in terms of making fewer mistakes when the prevalence of DVT was only 19%.
A score of 11 in the moderate risk category achieved a sensitivity of approximately 70% and a 100-Specificity of 30%. Two thirds of the population were deemed to have been correctly identified as true positives and developed DVT. Because sensitivity considered only those with DVT and specificity was those without DVT, the ROC plot was independent of the 19% DVT prevalence.

The area under the ROC curve for the above plot was calculated to be approximately 70%. This means that a randomly selected patient from the DVT group has a higher DVT risk assessment score than one from the group without DVT. However, it does not mean that a DVT occurs with a probability of 0.70 or that DVT is associated with a positive result 70% of the time.
Additionally, the result for the co-ordinates is statistically significant at < .001.
For the chosen threshold, the ROC also provided an unbiased estimate of the sensitivity and 1-specificity. This meant that the point neither overestimated nor underestimated the true value of sensitivity and specificity. The 95% Confidence Interval resided between 0.6-0.8.

Data extrapolation from Table 5.17 and the ROC curve (Figure 6) affirm that the score of 11 is the optimum cutoff point and reduces the amount of false negatives. However, lowering the cutoff value from □ 15 to 11 means that some patients identified as true positives will not develop DVT and thus increases the false positives. This situation is not problematic when non pharmacological and side effects free prophylaxes are applied. On the other hand, if heparin is given for the prevention of DVT, it may have some serious consequential effects. Heparin is associated with an increased risk of bleeding (approximately 5%) and Heparin Induced Thrombocytopenia Thrombosis (HITT) which occurs in approximately 1 % of cases (Warkentin et al, 1995). HITT characterised by low platelet count, haemorrhage and thrombi formation, causes serious adverse reactions and can be fatal (Anand et al, 1998). In such circumstances, clinicians must allow flexibility of

clinical judgement above the cutoff point of 11 in order to minimise the significant effect on false positives. In brief, the choice of the cutoff level relates to the relative importance of false positivity and false negativity for the condition (Gordis, 1996).

Likelihood ratios

Likelihood ratio is an alternative way of describing the performance of a diagnostic tool or a predictive index (Fletcher, Fletcher & Wagner, 1988). It is the ratio between the probability of a defined test result indicating the presence of a disease and the probability of a defined test result indicating the absence of a disease. Likelihood ratio summarises the same information as sensitivity and specificity but is more stable than sensitivity and specificity. This is because if the mix (low, moderate and high risk) of patients with DVT varies when their prevalence varies, sensitivity and specificity will change as well as predictive values. Because likelihood ratios can be generated for quite narrow slices of a test result, they will be less susceptible to such changes in the mix of patients with DVT (Sackett et al, 1991).

Likelihood ratio for the score values of the DVT scale was calculated for the following three reasons:

- First of all, it is independent of prevalence of DVT.
- Secondly, it offers the option of calculating all the four levels of cutoff score of the DVT assessment protocol and not just the absolute scores of ≥15 and ≤14.
- Thirdly, it subdivides and assigns patients to particular levels of measurement, thus resolving the problem of dealing with patients who are "outliers" to the levels of measurement.

To calculate the likelihood ratio of the DVT scale, the following formula was applied:

$$LR = \frac{TP/\langle TP+FN\rangle}{FP/\langle FP+TN\rangle}$$

Table 5.29 calculates the likelihood ratio for the four levels of measurement of the cutoff scores used for the performance of the DVT scale.

Table 5.29: Likelihood ratios for several levels of cutoff scores

	DVT Present		DVT Absent		
Cutoff range	Number	Proportion	Number	Proportion	Likelihood Ratio
≥15	7	7/28=0.25	12	12/120=0.1	0.25/0.1=2.5
11-14	12	12/28=0.428	25	25/120=0.208	0.428/0.208=2.0
7-10	5	5/28=0.178	45	45/120=0.375	0.178/0.375=0.47
≤6	4	4/28=0.142	38	38/120=0.316	0.142/0.316=0.44
Total	28		120		

Given the likelihood ratios are 2.0 and 2.5 for the cutoff scores of 11-14 and ≥15 respectively, probability of DVT increases when the patients have such score values.

The likelihood ratios for cutoff scores of ≤6 and 7-10 are 0.44 and 0.47 respectively and less than 1.0 and the probability of DVT declines.

The likelihood ratios for the four levels of cutoff scores lend support to the four score ranges of the risk assessment protocol.

The Positive Predictive Value (37%) of the DVT scale was largely influenced by the rather low prevalence of DVT reported. Importantly, the findings of the sensitivity and specificity of the DVT scale were also partially influenced by some uncontrollable factors in the study: efficacy of primary venous thromboprophylaxis and DVT preventative measures to combat venous stasis. Such preventative nursing interventions are leg elevation, application of graduated compression stockings, physiotherapy and early programme of ambulation. Graduated compression stockings reduce the incidence of DVT by approximately 68% (Wells et al, 1994). Data pooled for the sensitivity and specificity estimates were from patients across three directorates. In contrast to the low uptake of primary pharmacological venous thromboprophylaxis on the orthopaedic directorate, 52% of patients on the medical unit and almost 100% of the surgical patients were recipient of pharmacological intervention for DVT. Consequently, a large number of patients initially risk assessed and predicted true positive for DVT appropriately received venous thromboprophylaxis and became false

positive. As a result, the current moderate findings of the sensitivity and specificity of the DVT scale is likely to underestimate its predictive accuracy. It is no longer acceptable and ethical to plan case and control groups and withhold venous thromboprophylaxis in order to test the true sensitivity and specificity of a predictive index, when the efficacy of venous thromboprophylaxis has been proven beyond doubt (Kakkar & Lawrence, 1997). DVT and PE prophylaxis reduces the incidence by 50% or more (Vanek et al, 1991) and THRiFT (1998) recommends that prophylaxis should be mandatory in moderate and high risk groups.

Logistic regression

Logistic regression is a generalisation of the chi-squared test to examine the association of a binary-dependent variable with one or more independent variables (Campbell, 2001).

In order to predict the occurrence or non-occurrence of an episode of DVT, logistic regression analysis was applied. DVT as the state or dependent variable was dichotomous and took two values. If the patient developed DVT, it was coded 1 and 0 for no DVT.

Logistic regression was used to:

- investigate the relationship between the causal variables and a binary output variable, allowing for the confounding variables.
- find the independent factors that discriminated those who developed DVT from those who did not.
- provide a fast and effective way to obtain the estimate of probabilities of belonging to a specific population and the estimate of odds ratio of having a specific problem or condition (Yarandi & Simpson, 1991).
- develop prognostic indicators so those patients at risk could be duly identified so that the most appropriate venous thomboprophylaxis is administered.

To perform the logistic regression analysis, SPSS version 11 (2001) software programme was used for data collated on the three clinical directorates. The seven parameters of the DVT scale were entered for each patient. The trauma and surgical intervention parameters were constant for the patients on the medical ward, as was trauma to the surgical patients. Variables that were not applicable were removed from the analysis by default. The special risk category variables were not applicable to any of the 150 patients and were therefore useless as predictive indicators. The salient features of the SPSS output for logistic regression on the orthopaedic, medical and surgical directorates are illustrated in appendices 8, 9 &10 respectively.

Logistic regression like multiple regression allows variables to be included or excluded according to various algorithms and options. A forward variable selection procedure using the Wald Statistic was used, starting off with no

variables and adding variables if they were significant, stopping when no more significant ones were found. An equation was constructed using those variables found to be significant which computed the likelihood of developing DVT for given values of the significant variables.

The model for the data was also analysed for its goodness of fit statistic. It compared the observed probabilities to those predicted by the model. The probability of the observed results is known as the likelihood. Since the likelihood is a small number, less than 1, it is customary to use –2 times the log of the likelihood (-2LL) as a measure of how well the estimated model fits the data. A good model is one that results in a high likelihood (LL) function. This means a small value of –2LL. If the model fits perfectly, -2LL would be equal to 0.

To test the goodness of fit statistic, the Hosmer-Lemeshow statistic was applied to the data on the three clinical directorates. On the surgical unit, the chi-square statistic was 4.328 with 6 df with a p- value of .632 (Appendix 10) indicating that the null hypothesis was accepted and the model fits the data. The two R (residual) values ranged between 24% to 41%, quantifying the proportion of the variance explained by the model. Cox & Snell and Nagelkerke are estimates of the variance accounted for in the analysis.

Using the same test for the goodness of fit for the data, the chi-square was .001 with 1df and a p- value of .977 (Appendix 8) confirming that the data fit the model on the orthopaedic directorate. The variance was 24% to 34%. On the medical unit, the Hosmer–Lemeshow test recorded a chi-square of 3.698 with 7 df and a p- value of 0.814 suggesting that the data fit the model. Proportion of variance ranged from 17% to 25%. A nonsignificant goodness of fit test (.722) indicates that the data fit the model (Munro, 2001).

The classification of the table in logistic regression helps to assess the performance of the model by cross tabulating the observed response categories with the predicted response categories. Cells on the diagonal are correct prediction and cells off the diagonal represent incorrect prediction. The patients were considered to be in the predicted DVT group if the value of the predicted probability was greater than 0.5 or were considered

predicted non-DVT group otherwise. These predictions were compared to whether the patients had a DVT and the correct percentage given.

On the orthopaedic directorate 42 TN were correctly predicted and 1 TP was identified.

On the medical directorate, an overall 80 per cent of the patients were correctly predicted and classified. Only four of the twelve patients with DVT were correctly predicted (33.3 %).

On the surgical ward, an overall 90 per cent of the patients were correctly predicted. Of the TN, 41were correctly classified and 4TP were also correctly identified. 5 patients were misclassified (4 FP and 1FN).

The variables found to be significant were also given. The variables in the equation summarise the roles of the parameters in the model. ß is the estimated coefficient with Standard Error (SE). The ratio of ß to SE in column 2 and 3 respectively squared equals the Wald Statistic. Model parameters were deemed significant at the 5% level using the Wald Statistic. In logistic regression the ß- weights are used to determine the probability of a subject having DVT or not. Instead of a score, as with the continuous response linear regression, the logistic model reports on probability ranging from 0 to 1. If the probability is greater than 0.5, the patient would be predicted to have DVT. If it is less than 0.5, the individuals would be predicted not to have DVT.

The covariate *trauma* was found to be significant to the orthopaedic patients as *build* was to the medical patients and high risk diseases to the patients undergoing surgery. Interestingly, although DVT increases exponentially with advancing age, it was not found to be significant in all three clinical areas. On the other hand, high risk diseases were computed as a significant variable and presumably patients with advancing age developed the high risk diseases. Due to the number in the population being sampled, it must be pointed out that the analysis was not definitive. It was not possible to consider all the covariates of the scale as their applicability were restricted within the three directorates and with a limited number of patients considered. The required regression sample size depends on the desired

power, alpha and beta levels, number of predictors and the expected effect size. The simplest rule of thumb is $N \geq 50+8m$, when $N \geq 50$ is the minimum cases to each independent variable and m is the number of independent variables (Tabachnick & Fidells, 1996). Therefore, to achieve the desired power, α-level=0.05 and β= 0.80 for evaluation of the 7 independent DVT variables, 106 cases (50+ (8)(7)= 106) would be required and a total of 318 patients accrued for the three clinical directorates.
The analysis described has certain disadvantages. It has been performed in a series of patients and the results apply to this particular series only. Even if validity were tested and confirmed in another prospective similar series, it would not necessarily be applicable to a different group of patients in other clinical areas that were not tested because of the probable presence and absence of other known risk factors.

Although the logistic regression analysis discussed had some limitations, it was undertaken in the spirit of a preliminary investigative study and is a worthwhile exercise planning a prospective and a much larger study for testing the discrimination of the independent variables.
Logistic regression in SPSS also allowed for the data to be stored to construct a ROC curve for the independent variables (appendices 11, 12 & 13).

Practical application of the DVT scale

In order to evaluate the practical application of the DVT scale, a postal questionnaire survey was undertaken. Comments were invited from the users of the DVT scale and 22 out of 25 (88 %) DVT scale users responded. All the respondents were registered nurses with varying level of experience. Clinical experience ranged from less than six months for newly qualified staff to fifteen years of post registration experience for the senior practitioners. A skill mix of trained staff was represented: D grade staff nurse whose prime responsibility was to assess, implement and evaluate care delivery and F grade staff who led a team of E grade staff and below.
All respondents had no prior experience of using the DVT scale and this was considered to be important to the integrity of their responses. Any prior usage of the DVT scale could influence their clinical judgement and bias their eventual responses, in relation to its utility and practical application.

Detailed analyses of the questionnaires from the orthopaedic, medical and surgical directorates are located in appendices 14, 15 & 16 respectively. The feedback from the respondents are summarised below:

The DVT scale was very favourably evaluated across the three clinical directorates. Its design permitted its application and evaluation in all the three clinical specialities without necessitating any modification to the proforma. Items that were not applicable to a particular speciality were simply not rated. For example, the trauma risk category subscale was not applicable to the medical and general surgical directorates. Equally, covariates in the special risk category were not tested, as no patients were recruited with such clinical risk factors.
The DVT scale was described as promising, user friendly and easy to apply. There was good consensus that the cumulative effects of the risk factors enabled the respondents to configure a clinical risk profile based on multifactoral aetiology. Such clinimetry individualises DVT risk assessment in less than 3 minutes. It purported to complement clinical judgement and "reminds us of patients who otherwise might have been overlooked", thus making DVT assessment visible to demonstrate clinical effectiveness.

There was broad agreement that the DVT scale was a valuable teaching tool and could be readily used for education of staff and patient health promotion. It was generally acknowledged that the DVT scale had good practical potential as an audit tool, in terms of monitoring the incidence of DVT in patients and evaluation of their current venous thromboprophylaxis protocol. It has also been recognised that a formal assessment tool facilitates the audit cycle and improves the uptake of venous thromboprophylaxis (Byrne et al, 1996).

The following free text qualitative comments were recorded in relation to its strengths, limitations and recommendations for improvement:

Strengths

- "It gives a clear indication of who may be at risk".
- "It reminds you of patients who are vulnerable".
- Easy to apply in any area.
- Quick and self explanatory.
- Virtually self-explanatory.
- Covers all the categories.
- "It identifies patients at risk, who may not have been realised until doing the risk assessment".
- "Useful for patients who don't appear to be at risk at first glance".
- "It makes you take a second look for patients at risk".
- "It enhances clinical assessment, highlighting areas that may otherwise be missed".
- It makes the whole assessment process visible.

Limitations

- It does not differentiate between male and female.
- It does not recommend which prophylaxis to be used for moderate and high risk group.
- Needs a bit of background information with the scale and its application.
- Needs to get doctors on board so those high risk patients receive appropriate prophylaxis.
- Is myocardial infarction in the high risk disease category a previous MI or a current event?

Comments on improving the practical application of the scale

- Needs to be an integral part of patient assessment and not a stand-alone document.
- MI needs to imply previous medical history or current event.
- "Hope this tool become part of risk assessment in the same way the Waterlow has and we will quickly see an impact on patient care and standards".
- Can be integrated in a careplan already in use on the unit.
- It could be laid out in a more linear fashion as the Waterlow scale.
- Not really, all aspects of risks have been covered.

Discussion

Most of the limitations and comments in relation to the improvement of the DVT scale have been addressed in the concluding chapter of the study. However, the following three areas of concern also reported deserve some explanation as to their initial omission: sex difference

A daily risk assessment recording

Types of venous thromboprophylaxis

for each risk category.

Sex difference

Reportedly, the DVT scale did not differentiate between male and female patients, assuming that the females were the riskier of the sexes. Such assumption in terms of DVT occurrence is unfounded and presumably based on the understanding of the clinical tenet that female patients are at greater risk of developing pressure ulcer than males (Waterlow, 1985). Although Anderson et al (1985) reported a higher incidence of DVT in females in the Worcester study, they lived longer than their male counterparts and had additional DVT risk factors such as pregnancy and puerperium, HRT and oral contraceptives, which put them at greater risk. Coon (1978) and Nordstrom 91992) found no difference in the sex distribution of DVT.

It was also inferred that the lay out of the DVT risk calculator did not allow for daily risk assessment to alert changes in general condition of patients. Pressure sore risk assessment is undertaken daily for prompt detection of pressure ulcer. Although the Autar DVT scale looks deceptively like the Waterlow scale for pressure sore risk assessment, it is founded on the universally recognised Virchow's triad in the genesis of DVT and backed by best external evidence from researches and biosciences. Perhaps, the DVT scale should carry a warning that it is remotely a pressure sore risk calculator. Apart from changes in the level of mobility subscale, it is most unlikely that the other relatively permanent DVT risk factors of the subscale will change significantly on a day to day basis as to influence outcome. Significant thrombi development occurs as early as 24 hours of admission. It is for this reason that the International Consensus Statement (2001) recommends that DVT risk assessment be undertaken within this defined time frame, to enable prompt application of the most appropriate thromboprophylaxis.

Types of venous thromboprophylaxes for each risk category

Although the DVT scale identifies patients into a particular risk category based on the cumulative effect of the risk factors, reportedly it did not signpost the nature and type of prophylaxis required for each risk group. The Autar DVT scale (1997) was essentially as a data collection tool and for the purpose of evaluating the sensitivity of the tool was not designed to draw attention to any prophylaxis. However, the ultimate goal of any risk assessment tool is to guide clinical decision into the management of risk. The comment of the respondents was duly noted and the recommended DVT prophylaxis for each particular risk category now features prominently in the revalidated DVT scale (Figure 7: p187).

Available data were also analysed to audit the uptake of venous thromboprophylaxis strategies. The data collection tool (Autar, 1997) allowed for the recording of the types of prophylaxis prescribed for each patient. Data collated and analysed were used to compare and contrast current local thromboprophylaxis strategy with the recommendations of the international and national venous thromboprophylaxis consensus groups as previously outlined in table 1.5.

Data on venous thromboprophylaxis across the three clinical directorates are exhibited in table 5.30

Table 5.30: Venous thromboprophylaxis strategies on the three clinical directorates.

Types of venous thromboprophylaxis	Orthopaedics Frequency	Surgery Frequency	Medical Frequency
Leg elevation	4	0	0
Graduated Compression Stockings (GCS)	0	0	0
Intermittent Pneumatic Compression (IPC)	0	0	0
Unfractionated Heparin (UFH)	0	39	1
Low Molecular Weight Heparin (LMWH)	1	6	18
Unfractionated Heparin + Warfarin	0	2	4
Low Molecular Weight Heparin + Warfarin	0	1	1
Antiplatelets	1	0	2
No prophylaxis	44	1	24
Warfarin only	0	1	0
Total number of patients	50	50	50

The uptake of venous thromboembolism was strikingly low on the trauma/ orthopaedics, considering that the incidence of DVT ranges from 50-70 per cent in patients with hip fractures (Paiement & Mendelsohn, 1997). The International Consensus Statement (1997) recommends LMWH pre surgery and every 12 hours post-operatively or Warfarin within a therapeutic INR of 2.0-3.0 pre and post surgery.

Table 5.31 outlines the clinical characteristics of the coded patients with DVT on the orthopaedic directorate.

Table 5.31: Clinical data of the orthopaedic patients with DVT.

Patients'code	Age	Diagnosis	Prophylaxis	DVT destination
W100/22/c	92	Fracture neck of femur	Nil	Ward
W100/21/c	67	Fracture neck of femur	Nil	Home
W100/16/c	79	Ruptured patella tendon	Tinzaparin 4,500 units daily (post DVT)	Ward
W100/05/d	69	Infected right hip prosthesis	Nil	Home
W100/20/d	88	Fracture neck of right femur	Nil	Home
W100/21/d	74	Fractured right tibia and fibula	Nil	Ward
W100/24/d	89	Fracture neck of right femur	Nil	Ward
W100/23/d	90	Fracture neck of right femur	Nil	Ward

Five patients who were admitted for repair of hip fracture and who did not receive any known and proven venous thromboprophylaxis developed DVT. Leg elevation as a DVT prophylaxis was applied to four patients. Although leg elevation is simple, safe, increases blood velocity and mechanically prevents venous stasis (Ashby et al, 1995), there is insufficient data to substantiate its role in DVT prophylaxis when other alternatives already exist (Levine et al, 1996).

One patient was recipient of low dose aspirin. Opinion is divided into the efficacy of aspirin in DVT prevention. Antiplatelet Trialists Collaboration (1994) claims that antiplatelets such as aspirin reduces incidence of DVT by 37 per cent but others have reported that they are generally ineffective and convey no benefit in orthopaedic patients (Levine et al, 1996).

One patient was administered subcutaneous LMWH Tinzaparin 4,500 units daily to prevent recurrence of the problem and PE. Already established as effective therapy for DVT, Tinzaparin is also the first low molecular weight heparin to be licensed for the prevention and treatment of PE (Fegan, 1998).

In sharp contrast to the trauma/ orthopaedic directorate, a venous thromboprophylaxis strategy was in place on the surgical unit. Forty-nine

patients received some known prophylaxis and the one patient who was admitted for assessment and observation of abdominal pain did not and was discharged within 24 hours. It was not possible to follow up the patient for monitoring post discharge DVT, due to destination being unknown. As shown in table 5.23, a diversity of pharmacological prophylaxis was given but low dose heparin was the most popular choice. Thirty-nine patients received 5000 units of calcium heparin 12 hourly until they were ambulant. Although laboratory monitoring of heparin requires the estimation of Activated Partial Thromboplastin Time (APTT), no monitoring is necessary for low dose calcium heparin administered (BNF, 2000).

LMWH is as effective as low dose heparin in reducing the incidence of DVT (Kakkar et al, 1997). Six of the patients on the same directorate, but under the medical management of a different clinical consultant, were prescribed LMWH. Tinzaparin was given two hours before surgery subcutaneously followed by 3,500units every 24 hours for 7-10 days or until the patient was ambulant. LMWH has a number of advantages over conventional UFH. It reduces the risk of bleeding and the incidence of haematoma caused by LDH (Collins et al, 1988).

Interestingly, despite the risk of bleeding associated with heparin, no patients were prescribed any mechanical method of prophylaxis. Intermittent Pneumatic Compression and Graduated Compression Stockings have proved to be effective in reducing incidence of DVT (Wells et al, 1994) but seemingly, the consultants on the directorate were not convinced. IPC is not proven to prevent PE (Hull et al,1990) in general surgery, which heparin does.

Regrettably, eight patients (8/ 50=16%) who all received pharmacological venous thromboprophylaxis, developed DVT. The clinical characteristics of patients with DVT on the surgical directorate are outlined in table 5.32.

Table 5.32: Clinical characteristics of surgical patients with DVT.

Patients'code	Age	Diagnosis	Prophylaxis	DVT destination
W100/01s	71	Bowel malignancy	Sodium heparin	Home
W/ 100/02s	52	Epigastric pain	Tinzaparin	Ward
W 100/ 05s	70	EUA rectal biopsy	Sodium heparin	Home
W 100/16s	78	Bowel malignancy	Sodium heparin	Ward
W100/25 s	77	Bowel malignancy	Sodium heparin	Ward
W100/ 33 s	77	Bowel adhesions	Sodium heparin	Home
W100/ 39 s	77	Abdominal pain	Sodium heparin	Home
W 100/45 s	90	Bowel malignancy	Sodium heparin	Ward

This reported incidence of 16 per cent DVT is consistent with the 16.2 per cent frequency reported for a population of surgical patients in North America (Clagett et al, 1995). Two of the DVT patients developed the condition on the ward and were duly treated by standard secondary prophylaxis of sodium heparin infusion and titrated warfarin therapy. The other four patients developed DVT while convalescing at home and were duly referred to the anticoagulant clinic for treatment. While it is almost impossible for the whole population being studied to be DVT free, the 16 per cent DVT recorded on the surgical directorate could have been further reduced or halved by a combined regimen of venous thromboprophylaxis. It appears that general surgeons are yet to be convinced of the benefits of a combined regimen of venous thromboprophylaxis despite three studies on surgical patients confirming the efficacy of the combined pharmacological method with mechanical prevention (Tongren, 1980; Borrow & Goldson 1981; Wille Jorgenson et al, 1985) (Table 5.33).

Table 5.33: Combination regimens of prophylaxis in surgical patients

Study	LDH	LDH + GCS
Tongren (1980)	12/ 98= 12% DVT	4/98=4 % DVT
Borrow & Goldson (1981)	23/ 80= 26.7 % DVT	2/63= 3% DVT
Wille Jorgensen et al (1985)	12/102=12% DVT	2/94= 2% DVT

DVT does not exclusively affect orthopaedic and surgical patients and there are few prospective studies on incidence of DVT in medical patients. Kierkegaard et al (1987) reported an incidence range of 13-28 per cent in bedridden non-surgical patients. However, there are certain medical conditions that are associated with occurrence of DVT and PE. Additionally, medical patients who develop DVT usually present a combination of high risk factors such as advanced age and immobilisation, which increase their global risk.

Table 5.34 outlines the clinical characteristics of patients with DVT on the medical directorate.

Table 5.34: Clinical characteristics of the medical patients with DVT

Patient'code	Age	Diagnosis	Primary/ secondary prophylaxis	DVT destination
W100/02/29	48	Myocardial infarction	LMWH(Primary)	Home
W100/08/29	77	PE	UFH (Primary) Warfarin (Secondary)	Ward
W100/17/29	73	Myocardial infarction	UFH (Primary) Warfarin (Secondary)	Ward
W100/23/29	77	Left ventricular Failure	UFH (Primary) Warfarin (Secondary)	Ward
W100/24/29	78	Congestive cardiac failure	UFH (Primary) Warfarin (Secondary)	Ward
W100/25/29	78	PE	UFH (Primary) Warfarin (Secondary)	Ward.
W100/01/30	58	Left ventricular failure	None UFH & Warfarin (secondary)	Ward
W100/04/30	62	Myocardial infarction	None UFH & Warfarin (secondary)	Ward
W100/14/30	60	Chronic heart disease Previous PE	LMWH (Primary) UFH & Warfarin (Secondary)	Ward
W100/16/30	75	Post myocardial infarction	None	Home
W100/23/30	79	Congestive cardiac failure	None	Home
W100/24/30	82	Congestive cardiac failure	None	Home

In the sample of 50 medical patients, a DVT frequency of 24 percent (12/50) was reported and an overall, 48 per cent (24/50) did not receive any venous prophylaxis. Eight of the 12 patients developed DVT during their hospital stay and the other 4 had DVT at home as DVT remains a continuing problem after discharge (Scurr et al, 1988). It is obvious from table 5.34 that five patients, who did not receive prophylaxis, developed DVT. While DVT is preventable and prophylaxis reduces the incidence of DVT, it is never

reduced to an unachievable zero rate (Mol & Egberts, 1994). Although UFH was the preferred primary prophylaxis, 10 per cent of the patients developed DVT. This is quite high but broadly in agreement with the findings of Simmons et al, 1973 and McCarthy et al, 1977, noting that a fourfold increase is reported in control groups.

Thirty six per cent of the patients (18/50) received LMWH as primary prophylaxis but 11 per cent (2/18) developed DVT. All patients who developed DVT on the ward were treated with secondary prophylaxis to prevent recurrence of the condition and PE, its ultimate and fatal complication.

Antiplatelet therapy has shown to prevent DVT and PE in high risk medical patients and the Antiplatelet Trialists Collaboration (1994) recommend that low dose heparin is routinely used in patients with myocardial infarction and stroke. No patients in the study were recipients of antiplatelet therapy.

Overall, there is sufficient evidence to suggest that some venous thromboprophylaxis strategies are in place across the three clinical directorates, but were inconsistently applied. They do not measure up with the recommendations of the national and international consensus groups (International Consensus Statement 1997; THRiFT, 1998) who vigorously advocated the administration of DVT prophylaxis based on risk levels. Although all patients on the surgical directorate received pharmacological prophylaxis, it was indiscriminately administered, irrespective of risk levels.

Summary

Data from 148 patients were analysed to establish the consistency, sensitivity, specificity and predictive validity of the DVT scale. Venous thromboprophylaxis strategies were also scrutinised to evaluate the effectiveness of current protocol.

Five reliability studies were undertaken. A high total percentage agreement and kappa values ranging between 0.88 to 0.97 confirmed the consistency of the DVT scale. A DVT prevalence of 19 per cent (28 patients) was recorded. Using a cutoff score of ≥ 15, 115 patients (78%) were correctly predicted for outcomes and 33 patients (22%) were misclassified. 14 percent of patients who were incorrectly predicted developed DVT. For the optimal predictive accuracy of the DVT scale, a ROC curve was constructed. The co-ordinates of the curve indicated that a score of 11 captured nearly 70 per cent of patients predicted for DVT. However, the predictive ability of the prognostic index was deemed to be underestimated as the outcomes could have been influenced by uncontrollable factors such as the efficacy of venous thromboprophylaxis. A large number of patients, who were predicted to be at high risk, were recipient of venous thromboprophylaxis and as a result became false positives. Although there was evidence of venous thromboprophylaxis strategies, they were inconsistently and indiscriminately applied. DVT is a continuing problem and 39 per cent of patients (11/28) developed DVT after discharge from hospital.

Logistic regression analysis was also applied to data, but findings were not definitive due to insufficiency of data and lack of testability of some independent variables.

Finally, analysis of the postal questionnaire survey lent support to the value and practicable application of the DVT scale.

Chapter Six

Discussion, recommendations and conclusion

This final chapter reviews the analysed quantitative and qualitative data of the study and, based on the findings, evaluates the consistency, predictive validity and practical application of the Autar DVT risk assessment scale. The DVT risk calculator will be examined also in terms of its effectiveness in guiding clinical decision making for the primary prevention of deep vein thrombosis. In the light of the discussion of the findings, some changes to the clinimetric properties of the DVT scale will be recommended to enhance its overall performance as a risk calculator and prognostic index.

Reliability of the DVT scale

An assessment tool is of little value if it is inconsistently applied, even if it has a high sensitivity and specificity (Gordis, 1996). Reliability and validity are both necessary conditions (Gibbon, 1995) and it is within such a context that the DVT scale will be judged.

High total percentage agreement and excellent kappa values recorded in the five reliability studies and discussed in chapter five confirm the consistency of the DVT scale. The findings indicate some modification to enhance reliability of the scale further. Nursing assessment instruments often get misconstrued in their application due to unavailability of the original information to understand the theory underpinning them. Such a misunderstanding results in inter rater disagreement. It is extremely important to ensure that potential users of an instrument have a good understanding of the underpinning theory as well as access to the original references for familiarisation with the tool (Cormack & Reynolds, 1992). The relevant publications on the DVT scale were made available to the ward staff where the DVT scale in this study was tested and this enabled them to access the literature as and when needed. Additionally, regular visits to the clinical areas by the researcher to monitor the research activity allow for dialogues and clarification of issues with the nurse participants. Despite the high consistency of the DVT scale as substantiated by the recorded measure of agreement, there is no room for complacency and higher

reliability was achieved by responding appropriately to the feedback from the postal questionnaires. Some problematic issues in terms of the clarity of some variables were reported. It was noted that some assessors on the medical directorate were uncertain as to when the variable "myocardial infarction" as a risk factor applied to their patients. Acute myocardial infarction is one of the leading causes of admission to the Medical Assessment Unit (MAU) and incidence of DVT ranges from 20-40 per cent in the acutely ill patients with this serious clinical condition (Carter et al, 1987). Although a previous history of DVT increases the likelihood of its recurrence sevenfold (Samama et al, 1993), currently available data do not support the assumption that a previous history of myocardial infarction makes patients vulnerable to DVT. Recent advances in coronary care have revolutionised the management of myocardial infarction and a high recovery rate has been recorded (Jowett & Thompson, 1989). Most patients with myocardial infarction go on to have a normal life after a period of rehabilitation (Lindsay & Gaw, 1997).

As the variable "myocardial infarction" is ambiguous and is inclined to lead to a bias in its interpretation as either an acute episode or a past medical history of the patient, it is recommended that the clinical variable be redefined. The term "acute myocardial infarction" (Figure 7) is preferred and serves to emphasise an acute attack of event necessitating hospital admission and its immediate potential for causing DVT.

AUTAR DVT RISK ASSESSMENT SCALE REVALIDATED (2002)

Name:

Unit No:

Ward:

Age:

Type of admission:

Diagnosis

AGE SPECIFIC GROUP (years)	score
10-30	0
31-40	1
41-50	2
51-60	3
61-70	4
71+	5

BUILD / BODY MASS INDEX (BMI)
Wt(kg/ Ht $(m)^2$

Build	BMI	score
Underweight	16-18	0
Average/ Desirable	20-25	1
Overweight	26-30	2
Obese	31-40	3
Very obese (morbid)	41+	4

MOBILITY	score
Ambulant	0
Limited (uses aids, self)	1
Very limited (needs helps)	2
Chair bound	3
Complete bed rest	4

SPECIAL RISK CATEGORY	score
Oral Contraceptives:	
20-35 years	1
35+ years	2
Hormone replacement therapy	2
Pregnancy/ puerperium	3
Thrombophilia	4

TRAUMA RISK CATEGORY *Score item(s)* ***only preoperatively.***	score
Head injury	1
Chest injury	1
Spinal injury	2
Pelvic injury	3
Lower limb injury	4

SURGICAL INTERVENTION: *Score only one appropriate surgical intervention.*	score
Minor surgery < 30 mins	1
Planned major surgery	2
Emergency major surgery	3
Thoracic	3
Gynaecological	3
Abdominal	3
Urological	3
Neurosurgical	3
Orthopaedic (below waist)	4

CURRENT HIGH RISK DISEASES: *Score the appropriate item(s)*	score
Ulcerative colitis	1
Polycythaemia	2
Varicose veins	3
Chronic heart disease	3
Acute myocardial infarction	4
Malignancy (active cancer)	5
Cerebrovascular accident	6
Previous DVT	7

ASSESSMENT INSTRUCTION
Complete within 24 hours of admission

Scoring: Ring out the appropriate item(s) from each box, add score and record total below;

Total score:

Assessor:

Date:

ASSESSMENT PROTOCOL

Score range	Risk categories
≤ 10	**Low risk**
11-14	**Moderate risk**
15 ≥	**High risk**

Please record any other clinical observations that may supplement this DVT risk assessment.

VENOUS THROMBOPROPHYLAXIS

Low risk: Ambulation+ Graduated Compression Stockings.
Moderate risk: Graduated Compression stockings+ Heparin + Intermittent Pneumatic Compression Stockings.
High risk: Graduated Compression Stockings+ Heparin+ Intermittent Pneumatic Compression.

International Consensus Group recommendation, 2001

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The consistency of any risk assessment tool can be maximised by careful design, simple and clear instruction (Gibbon, 1998). A problem was identified in this area despite a clear and simple instruction. In the DVT risk assessment of patients, the assessors were clearly briefed and instructed to record the trauma risk category of the DVT scale only preoperatively. This variable is no longer applicable if the subjects undergo surgery for therapeutic management of the trauma within 24 hours of admission. However, despite the explanation and the italicised instruction (figure 3), a few assessors continued to record the trauma risk factor and the surgical intervention simultaneously for some subjects. It can only be assumed that their action was automatic and the response, accidental. As a result, some patients received double risk weighting, which inflated their aggregate score and overstated the degree of risk. This human oversight can be resolved by writing the DVT scale into a user friendly computer software programme. This means that the assessors will be asked to choose only one most appropriate option between the trauma risk factor and the surgical intervention category. Any attempt to score both categories will not be permitted and the assessor not allowed to progress to the next stage to complete the assessment process until the computer prompt has been executed.

In the interim, it is anticipated that the new layout of the DVT risk assessment chart (Figure 7) designed as a risk calculator and not a data collection tool will address this misunderstanding.

The surgical intervention subscale is also an area where additional specific information can enhance the consistency of the scale. Orthopaedic surgery as an independent risk factor carries a high weighted score of 4 relative to the other types of surgery in this subscale. Incidence of DVT in orthopaedic patients ranges from 50 to 75 per cent (Das, 1994). Manipulation of the leg causes distortion and occlusion of the femoral and popliteal veins and is claimed to cause DVT (Stamatakis et al, 1977). However, this high incidence of DVT in orthopaedics relates only to patients undergoing below waist surgery such as total hip and total knee arthroplasties and surgical repair of fractured femur and tibia and fibula (Geerts et al, 1994). Incidence of DVT for other orthopaedic procedures such as upper limb surgery is not

proportional to lower limb surgery. For this reason, it is recommended that the variable "orthopaedic" be more explicitly redefined to denote below waist orthopaedic surgery (figure 7) and this will serve to differentiate this from other orthopaedic surgical procedures carrying little or no risk of DVT.

In the same subscale, to make the distinction between major surgery and emergency major surgery further, the former is redefined as "planned major surgery", to highlight the difference in risk. Elective major surgery carries an incidence range of 0.2 to 2.2 per cent rising to 2.7 per cent for major emergency surgery (Kakkar et al, 1970, Coon, 1976).

Predictive validity

The DVT risk assessment scale correctly predicted and classified 78 per cent of patients. Sensitivity and specificity are inversely related (Mausner & Kramer, 1985).
108 patients were correctly predicted not to be at risk and did not develop DVT. A high negative predictive specificity of 90 per cent was achieved against a low positive predictive value of 25 per cent (Table 5.12). A high predictive value of 84 per cent of negative outcomes was also achieved at the cost of a predictive value of 37 per cent of positive outcomes (table 5.16) and was consistent with the caveat that:

"As prevalence falls, positive predictive value must fall alongside and negative predictive value must rise"
Sackett et al, 1991: p88.

Testing and refining an instrument to augment its predictive validity is an iterative process and needs ongoing evaluation (Priest et al, 1995).
DVT prophylaxis or intervention masks the efficiency of any risk assessment scale and the higher the quality of intervention, the least well risk assessment scales perform (Deek, 1999). In view of interventions, risk assessment scales must not be seen in terms of absolute risk measurement but as conditional risk (Lyne, 2000).Future validation of the DVT scale must continue to focus on those risk factors, old and new alike associated with DVT and their inclusion or exemption based on compelling evidence.

Some of the subscales of the DVT risk calculator will need to be revisited and recommendations made accordingly, in order to enhance its predictive ability.

Age specific group

Great Britain is an ageing society. Since the early 1930's, the number of people aged over 65 has more than doubled and today a fifth of the population is over 60. Between 1995 and 2025, it is estimated that the number of people over the age of 80 is set to increase by almost a half and the number of people over 90 will double (DoH, 2001).

DVT increases exponentially with advancing age (Rosendall, 1997). People are living longer. Older people now comprise two thirds of all patients in acute hospital settings, partly due to the demographic shift but also due to lack of alternative settings for health and social care (DoH, 2001).

A DVT rise of 20 per cent is reported in the 40-60 year old patients. This doubles between the age of 60 and 70 years and in patients over 70 the figure trebles (Borrow & Goldson,1981; Caprini & Natonson, 1989). This exponential rise is further substantiated by the Office Population of Censuses and Surveys (OPCS, 1990) which reported 205 recorded cases per 1000 of DVT in the 75-79 age group compared to 123 cases in the 70-74 group and 100 in the 65-69 years old. Data analysis on the medical directorate also noted an increase in the number of elderly patients admitted in their 70's and 80's who are the most vulnerable group. Despite an overall low aggregate risk score placing them in the no risk category, two medical patients in this category developed DVT and advancing age was the only independent risk factor. In the age specific subscale, the 51-60 and 61+ age groups were assigned respective scores of 3 and 4. Relative to the incidence of DVT that rises sharply in the different age specific groups, it is judged that elderly patients in the 70-80 age group should be recognised as a higher risk group and therefore be assigned a risk score of 5.

Hormone Replacement Therapy (HRT)

Opinion has been divided on the association between Hormone Replacement Therapy (HRT) and the occurrence of venous thromboembolic diseases (THRiFT, 1998). At the time of the pilot study (Autar, 1994) and the launch of this investigation (Autar, 1997), the causal association between HRT and DVT was inconclusive and questionable. Evidence from earlier studies did not lend support to an increased risk of DVT or PE among HRT users (Boston Collaborative Drug Surveillance 1974; Moore, 1976). Notelowitz & Ware (1982) and Carter (1992) also found no association between HRT and DVT. Based on insufficiency of evidence available at that particular time, HRT as a risk factor was not considered in the development of the DVT scale.

However, two recent studies (Daly et al, 1996 and Jick et al, 1996) have demonstrated a positive causal relationship between HRT and DVT and the estimated relative risk was 3.5 and 3.6 respectively. Perez Gutthann et al (1997) conducted a case control study and concluded an overall twofold relative risk of venous thromboembolic diseases associated with the current use of HRT. Most recently, a randomised controlled trial (Lowe et al, 2000) confirmed a two to fourfold increase of DVT rate in women taking HRT.

The benefits of HRT outweigh its potential side effects (Cook et al, 2001). However, there is now overwhelming evidence from the most recent studies to conclude that a causal relationship between HRT and DVT exists. As a result, HRT is now considered as a risk factor in the revalidation of the DVT scale (Autar, 2002). As illustrated in the re-validated scale (figure 7), HRT is a new addition to the special risk subscale and is assigned a risk score of 2.

Thrombophilia

Thrombophilia or Hereditary Thrombotic Disease (HTD) represents a group of abnormalities in which patients have recurrent thrombotic events because of genetic defect(s). At least nine abnormalities have been confirmed but in 60-70 per cent of cases the cause of thrombophilia are still unknown (Marlar: in Wood & Burn, 1994). As a new condition is confirmed, risk

assessment followed by venous thromboprophylaxis must be seriously considered. To reflect this change, thrombophilia as a hypercoagulable state and independent risk factor is included in the special risk category subscale. 90-95 per cent of patients with thrombophilia present with DVT (Marlar & Mastovich, 1990) and proportional to the degree of risk relative to other risk factors in this subscale, it is assigned at a risk score of 4

Air travel as a risk factor

Long-haul flights and air travel in general as causative of DVT have been a subject of much controversy. As a result of recent press publicity, audiences at workshops, conferences and study days have often challenged the researcher in relation to the omission of air travel as a risk factor for DVT.

First of all, to justify this omission, it is important to put air travel into some perspective regarding the risk. Most reports published are narratives and the estimated risk in terms of percentage of person trips affected is unquantifiable and marginal. Most of the reports are derived from small numbers of air passengers and they can be criticised on account of their inaccuracy due to the retrospective analysis of the insufficient data. There appears to be an individual susceptibility to DVT. The passengers who developed DVT had other "patient-related" risk factors such as advanced age, previous DVT, chronic illnesses, malignancy, hormone therapy and recent lower limb injury (Eklof et a, 1996). These well-recognised risk factors have a cumulative effect (Autar,1996a) and are already well represented in the DVT scale. Such an argument is also in agreement with the "multiple hit theory" of venous thromboembolism postulated by Rosendaal (1997) that several factors combine to provoke an acute episode of DVT or PE in an individual. Co-morbid features such as obesity and varicose veins are often present in those passengers who developed DVT (Tardy et al, 1993).

Seven "cabin –related" risk factors have also been implicated, linking air travel with DVT (Eckloff, 1996). Traditional narrative review articles are frequently misleading and therefore unreliable for evidence based decision making (Antmann et al, 1992). While current evidence on air travel as a risk factor is anecdotal and weak, there is strong consensus that immobility rather than the environment is the significant factor (Cook, 2001). A

comparison of "First Class" with "Economy Class" passengers should make a revealing study.

High risk diseases

The high risk disease subscale is an area of risk assessment that needs to be critically reviewed. In the light of recently available evidence or lack of it, some of the high risk diseases need to be re-examined and any change warranted duly justified.

Specifically, clinical conditions such as sickle cell anaemia, haemolytic anaemia, varicose veins and cerebrovascular accident as risk factors for DVT need to be re-evaluated.

Haematological conditions

Pearson & Wetherby-Mein (1978) reported a positive correlation between DVT and polycythaemia. The Polycythaemia Study Group (PSG) has confirmed a high incidence of DVT in patients over the age of 70 (Wasserman et al, 1981) and hypercoagulability in Virchow's triad is the most prominent clinical pathophysiology. It is therefore sensible to retain polycythaemia as a risk factor with its original risk score of 2.

On the other hand, current data and evidence on sickle cell anaemia and haemolytic anaemia do not lend any conviction to the view that they are risk factors in the genesis of DVT. Both sickle cell anaemia and haemolytic anaemia have been implicated in earlier literature for the reason of restricted blood flow and the release of cell breakdown products. In sickle cell anaemia thrombi occur frequently in the microcirculation, resulting in infarction in the lung tissue, spleen, kidneys and bone. There is no recorded evidence that it affects the macrocirculation and causes PE, secondary to proximal and distal DVT (Bell & Simon, 1982).

Haemolytic anaemia is a premature accelerated destruction of erythrocytes and has been previously implicated as a risk factor. While haemolytic anaemia causes jaundice due to haemolysis, incidence of DVT is relatively rare and there is no classified data to support any direct causal association with DVT (Belcher, 1993). It appears that early publications linking sickle cell

anaemia and haemolytic anaemia to DVT, due to increased blood viscosity may have been speculative and exaggerated. A paucity of literature and unavailability of any classified hard and recorded data do not substantiate any causal association with DVT (Serjeant, 1992). In the absence of classified data and no new evidence, it is highly unlikely that they will contribute to the predictive accuracy of the DVT scale. It is therefore reasonable to recommend that the two items be deleted from the subscale (Figure 7).

Varicose veins

The positive association between varicose veins and frequency of DVT is not a matter of controversy *per se*. Consistent with its high ranking by the European Consensus Group (1991) and THRiFT (1992), varicose veins as a DVT risk factor features prominently in the high risk disease subscale. It is associated with a high risk score of 6, second only to previous DVT and CVA (Autar, 1994; Autar, 1997).

Recent debate on varicose vein as a covariate in the genesis of DVT is not disputed but is challenged in relation to the nature of its association. An association does not necessarily imply a direct causation (Norrell, 1995). Associated risk factors combine to exert an additive effect, culminating into an episode. On the other hand, independent risk factors are discriminative markers, acting singly to provoke an acute event (Nicolaides & Irving, 1975).

Clinical opinion is divided about the direct relationship between varicose veins and DVT occurrence. Several investigators have utilised varicose veins as an independent covariate in their predictive indices to identify patients at risk of DVT (Nicolaides & Irving, 1975; Clayton et al 1976; Crandon et al, 1980; Lowe et al 1982). Many vascular surgeons (Campbell & Riddler, 1995; Campbell,1996) are now challenging this clinical view. They claim the assumption that DVT is an independent risk factor is derived from a lack of understanding of the differences between the deep and superficial veins of the lower limbs. This scepticism is evident in a survey of venous thromboprophylaxis for varicose veins surgery. Only 29 per cent of vascular surgeons consider varicose veins as a high risk covariate, necessitating primary prevention (Campbell & Ridler, 1995).

Current literature on varicose veins is controversial and does not provide specific data on the frequency of DVT, primarily to the presence of varicose veins. The studies showing a relationship between DVT and varicose veins were undertaken in patients who had major abdominal surgery and invited criticism for the population sample also contained older and obese patients. Varicose veins may have coexisted incidentally with major abdominal surgery, advancing age and obesity as additive factors to cause DVT. Hirsh & Hoake (1996) and Gorman et al (2000) accept that varicose veins increase the risk of DVT but concede that some more definitive work need to be done in this area. In a study of 1231 patients, Anderson and Wheeler (1995) reported a DVT incidence of only 5.8 per cent due to varicose veins.

Most patients with varicose veins belong to a group of population with advancing age and limited mobility who may also be affected by leg ulcers, secondary to the complication of varicose vein (Callum, 1992).

Future studies must take into account other covariates in patients are discriminated from varicose vein in the pathogenesis of DVT.

In the interim, retaining the high risk score of 6 for varicose veins is unjustified because it overpredicts the risk and increases the false positive rate. In conclusion as there is little evidence currently to support varicose veins as an independent risk factor (Campbell, 1996; Agu, 1999), the associated role of varicose vein in predisposing to DVT should be acknowledged, but its original risk score is halved (Figure 7).

CVA and previous DVT

CVA and a previous DVT are very well recognised high risk diseases in the causation of DVT, each associated with a risk score of 7 (Autar, 1994; Autar, 1997). The incidence of DVT ranges between 42-60 per cent for CVA (Kamal 1987; Brunner & Suddarth, 1992). In patients with a previous history of DVT, the recurrence of an episode is reported to be between 48-68 per cent (Kakkar et al 1970; Dalen et al, 1986). An even higher risk than CVA was reported by Samama et al (1993) who recorded an odds ratio of 7.9 for patients with previous DVT. There is now a general consensus that previous DVT predisposes to the recurrence of the condition and is the highest risk

factor in the causation of DVT (Nordstrom et al, 1992; Samama et al, 1993; Anderson et al, 1995).

As the risk of DVT is higher for patients with previous DVT than with CVA, previous DVT is capped at the risk score of 7 and revised risk score of 6 assigned to CVA. In this way, a small difference in the risk associated with these conditions is maintained.

The DVT risk assessment protocol

Evaluation of the sensitivity and specificity of the DVT scale indicates that some minor modification to its assessment proforma would enhance the predictive accuracy of the instrument. The current DVT risk assessment strategy (Autar, 1997) requires the known clinical risk factors for the patients to be identified. This places them into one of the following risk categories: no risk, low, moderate and high risk. It is important to identify those at risk and differentiate them from those who are not so that the limited resources could be targeted most effectively. That is why a no risk category was incorporated into the assessment protocol.

DVT is a disease of the hospitalised patients (Douglas, 1978) and in reality all patients, by virtue of admission for investigation or treatment should be regarded as at risk. This was evident in the data analysis for the medical directorate. Two patients on the medical ward developed DVT despite having a low score of less than 6, which placed them into the no risk category. With an increasing number of claims being made for clinical mismanagement (NAHAT, 1991) it is prudent to err on the side of caution and assume all patients are at risk. A DVT incidence of 11 % for patients with no risk factors present and deemed not to be at risk, was reported by Anderson & Wheeler (1995; Table 3.16). It is therefore recommended that the four risk categories of the DVT scale be reviewed to merge into three distinct risk categories (figure 7). The removal of the no risk category from the assessment protocol places the low risk category into a wider risk score range of less than 10 and resolves any problem of spurious precision between the two risk categories. The other risk score ranges of 11-14 and 15 and over remain unchanged and identify the moderate and high risk categories respectively. Modification of the assessment protocol into three

risk categories is also consistent with the recommended assessment strategy by the national and international consensus groups (European Consensus Group, 1991; International Consensus Group, 1997; THRiFT, 1998).

Collapsing the no risk category into the low risk category rules in a total of 92 patients and yields a DVT incidence of 10% (9/92) for the low risk category. This is consistent with previous DVT occurrences reported by Salzman & Hirsh (1982) and THRIFT(1998). An absolute DVT incidence of 32% (12/37) reported in this study, is compatible with the generally recorded incidence range of 11-40 % for the moderate risk category (International Consensus Statement,1997). Although the incidence of DVT ranges between 40-80 % for high risk category, in this study a 37% (7/19) occurrence is noted and this is primarily due to fewer patients in the high risk group being recruited. Table 6.1 exhibits the DVT incidence for the three risk categories of the modified risk assessment protocol.

Table 6.1: DVT incidence for the three risk categories.

Risk categories	No of patients in risk category	No of DVT recorded	DVT (%) recorded
Low	92	9	10
Moderate	37	12	32
High	19	7	37
Total	148	28	19

Practical application of the scale

The DVT risk assessment scale has achieved some promising results in terms of consistency and overall correct percentage prediction. It is reproducible, easy to use, comprehensive and takes less than 3 minutes to complete. It can be universally applied to clinical areas, as all proven and known clinical risk factors in the pathogenesis of DVT have been included. The special risk category subscale is represented by specific variables such as oral contraceptive and oestrogen therapy, pregnancy and puerperium and thrombophilia. Due to the special nature of the risk factors present in a particular patient population, the testability of the items in this subscale was

not possible. None of these variables were applicable to the patients in the study and were recorded as constant in the logistic regression analysis. Patients who were taking oral contraceptive therapy were advised to stop the contraception three weeks prior to the planned surgery to minimise risk of venous thromboembolism, as recommended by the British Medical Association (BMA) and the Royal Pharmaceutical Society of Great Britain (BNF, 1994). No patients receiving HRT or sufferers of thrombophilia were admitted. Further to test the validity of the items in the special risk category, it is suggested that such populations will be targeted in future studies. In particular, validating the DVT scale on the obstetric speciality will enable the testability of pregnancy and puerperium as a high risk factor.

DVT scale as a teaching tool

It is generally recognised that the DVT scale is also a valuable teaching tool as evident in the evaluative responses of the clinicians to the postal questionnaires (appendices 14,15 & 16). Other risk assessment instruments (Arcelus et al 1991; Caprini et al 1991; Bahal & Silverman et al, 1993) have all utilised the known risk factors to assess patients for risk of DVT. While this approach classifies patients into risk categories according to the number of risk factors present, it provides no basis for comparison of the risk for individual patients and explaining why some individuals are more at risk than others. On the other hand, the very design of each subscale puts the assessment process in a risk perspective and explains why an individual is or is not at risk. The covariates making up each subscale illustrate the low or absent risk relative to the highest risk factor, as quantified by the risk score assigned for each item. The assessment protocol is self explanatory, as a low risk score denotes a low level of risk and the assignment of a high score identifies a high level of risk.

The DVT risk assessment tool has already been incorporated into the spiral nurse education curriculum for students undertaking programmes, leading to BSc in Nursing and Diploma in Nursing awards, at the local university. As DVT is a problem common to all the clinical specialities, the DVT risk calculator is used as a tool for teaching the primary prophylactic and secondary crisis intervention management of the condition. In the current

curriculum, the DVT scale is first introduced to explain the thrombogenic mechanisms (Table 3.16) underpinning Virchow's triad in the pathophysiology of DVT. This introduction is then followed by a problem solving approach learning strategy to evaluate the learning outcomes. The students are presented with case studies and using the DVT scale, they are required to work out the assessment process and place each patient into an appropriate risk category.

Staff preparation is critical to the successful development an Implementation of any protocol. Current literature review suggests that there is no publication with regard to the evaluation of nurses' knowledge of venous thromboembolism and the DVT scale can potentially fulfil two functions. First of all, risk assessment lies at the very core of a venous thromboprophylaxis protocol and the DVT risk scale is developed for this purpose. Secondly, it can be applied to evaluate nurse's knowledge of DVT and ultimately prepare them for implementation of the protocol.

The DVT scale can also be used to train anticoagulant nurse specialists, practice nurses and those undertaking the formal community nurse education programme. They are becoming increasingly involved in the primary and secondary venous thromboprophylaxis management of DVT in their integrated caseload. Historically, patients with DVT have been treated initially by Unfractionated Heparin (UFH), necessitating inpatient care. But with the advent of Low Molecular Weight Heparin (LMWH), all these patients are now managed effectively by domiciliary nursing services and outpatient anticoagulant clinics. The introduction of LMWH has revolutionised the management of DVT and their benefits are listed below:

- It needs only be administered subcutaneously and eliminates the need for infusion equipments and monitoring and saves staff time (Fegan, 1998).
- A single dose daily provides anticoagulation for 24 hours (Friedal & Balfour, 1994).

- It removes the need for extensive laboratory monitoring. Clotting tests as Prothrombin Time (PT) and Activated Partial Thromboplastin Time

(APTT), which are regularly required for UFH, are not required (Clagett et al, 1992).

- It reduces the adverse high rate of wound haematoma caused by UFH (Collin et al, 1988).

- Incidence of DVT is 5.3 per cent for LMWH compared to 6.7 per cent for UFH (Perkins & Galland, 1999).

- Managing adverse outcome of LMWH costs £144.75 compared to £ 195.50 for UFH (Valette et al 1991).
- Patients who receive LMWH spend a mean of 1.1 days in hospital as compared with 6.5 days for standard heparin (Levine et al, 1996).

It is recognised that with the appropriate training, nurse specialists, practice nurses and community nurses can run anticoagulant clinics as well as haematologists do (Van de Pette & Mackie, 1996). Nurse-led anticoagulant clinics (Brown et al, 1999) and Community Nurse-led outpatient clinics (Deagle, 1998) for DVT management are evolving rapidly to meet the needs of the health care. More patients are being treated at home, and the Southampton University Trust Hospitals (SUTH) initiative described in chapter one, is an example of effective care delivery at the point of demand to benefit patients and reduce hospital cost.

DVT is a continuing problem and patients often develop thromboembolic complications up to six weeks following discharge from hospitals (Scurr et al 1988). The old cliché that DVT is a disease of the hospitals (Douglas 1978, Grace, 1993, Anderson et al, 1995) does not ring entirely true. Nilsson et al (1997) reported a DVT incidence of 38 per cent occurring at home in patients whose prophylaxis was restricted only to their hospital stay. The report is further supported by the findings this study. Eleven out of the 28 patients with DVT (39%) developed the condition at home: none of them were recipient of any prophylaxis post hospital discharge. There is now an inverse relationship between the duration of hospitalisation and thromboembolic complications, due to early discharge (Haas, 1997). With increasing demand for hospital beds for acute admission, patients are being discharged home earlier. Risk factors persist beyond hospitalisation and community nurses are well placed to assess the patients with the DVT scale

and monitor them for any venous thromboembolic events. One fourth of PE occurs after discharge home (Huber et al, 1992) and registered nurses must:

"Safeguard and protect the interests of individual patients and clients" (UKCC, 1992) and
"act to identify and minimise the risk to patients and clients" Nursing and Midwifery Council (NMC, April 2002).
Having applied the DVT scale to assess risk, the community nurses can also take appropriate actions to minimise them and prevent DVT. Although the prescription of pharmacological prophylaxis is the province of physicians, nurses can actively initiate side-effects free and safe mechanical prophylaxis. They can recommend and encourage patients to wear graduated compression stockings to promote venous return. In a meta-analysis, Wells et al (1994) reported a 68 per cent overall risk reduction for prophylaxis with graduated compression stocking. Other methods of mechanical prophylaxis can be achieved by a health promotion programme aimed at encouraging active and passive exercises and leg elevation. Active and passive exercises prevent stasis by promoting venous valve function and muscle action pump (Cotton & Roberts, 1977). A 10-degree elevation of lower limbs effects a 30 per cent increase in blood velocity (Wolfe & Sarbiston, 1980). A more recent study by Ashby et al (1995) confirms that leg elevation of around 6 degrees in the supine position aids blood flow and is a simple yet very effective prophylaxis against DVT.

Decision making process

Evidence from audit of venous thromboprophylaxis across the three clinical directorates for this study, suggests that it is inconsistently applied due to lack of organised strategies to guide the decision making process. In the absence of any venous thromboprophylaxis protocol in place, uptake is alarmingly weak on the trauma/orthopaedic unit (12%) and only moderate (48%) on the medical directorate in one NHS trust. A DVT incidence of 20% was recorded. On the other hand, although there were no documented venous thromboprophylaxis strategies, 98%(48/50) of the patients on the surgical directorate of another NHS trust received some proven form of prophylaxis.

SIGN (1995) and THRiFT (1998) have strongly recommended protocol development to facilitate the uptake of the most effective prophylaxis, based on risk assessment. Just as assessment is the initial stage of the nursing process (Crow, 1977), DVT risk assessment is at the very foundation of protocol development. DVT risk assessment predicts risk and enables the clinicians to make professional judgement as to what is the most effective prophylaxis. Although prediction is not always accurate, it serves to guide action to benefit patients.

Simms & Fought (1989) state that decision making is a blend of theory and expertise. The more experience the practitioner has, the better is the nature of clinical judgement to guide decision-making. Benner (1984) describes expertise as being generated from concrete past experience. Experience

"does not refer to the mere passage of time or longevity. Rather it is the refinement of preconceived notions and theory through encounters with many actual situations".
Benner,1984.

The novice, on the other hand, does not have the ability to reflect on past experience and Benner (1984) suggests that expertise and knowledge can only be gained by direct encounter with an event. Experience appears fundamental to explaining the concepts of "art" and "science" of clinical decision making. This may have implications for future training, which can be developed to include an apprenticeship component, with the necessary supervision within a clinical setting. As it happened, all the data collectors in the study were registered nurses with good clinical background and experience in the management of thromboembolic diseases.

In order to remind clinicians about the internationally and nationally recommended venous thromboprophylaxis for each of the three risk groups, they are now printed on the DVT scale chart (Autar, 2002 : p192). A formal DVT risk assessment chart promotes the uptake of venous thromboprophylaxis (Bahal & Silverman 1993; Byrne et al, 1996). Thus far, the DVT scale has focused primarily on the risk calculation and future studies should audit the impact of this instrument in making decision about the choice of prophylaxis.

Conclusion

The DVT scale has yielded some promising results and attracted considerable interest both nationally and internationally. It has good practical application and the theory underpinning it is knowledge embedded and developed in clinical practice. The DVT scale is not set in tablets of stone and nurses must not be complacent in its application but should exercise their professional and clinical judgement to complement it. For example to put this into a clinical perspective, some high risk patients with a score of □ 15 develop DVT while others with the tied score do not. Those who do not are often patients whose mental status enables them to co-operate in care to minimise DVT risk. Although they are immobile, they understand simple nursing instruction to engage in antistasis exercise such as flexion and dorsiflexion of the foot to activate the muscle pump. In such clinical situations, while recognising the risk to patients and acknowledging that mental status is not a known DVT risk factor, using their professional judgement adjunctively with the DVT scale, can make a difference. Capitalising on the co-operation of the patients has enabled them to reduce the risk associated with immobility. The new design of the revalidated scale should encourage clinicians to record such relevant observations based on professional judgement to supplement the DVT risk calculator.

The goal of this study has been to obtain a large enough sample for statistical significance yet expedient and economical at the same time. *A priori* sample size was estimated using the G* Power computer software and for a low to medium effect sizes of 0.23, 149 patients were required. Cohen (1977) concedes that for a new area of research enquiry such as the evaluation of the DVT scale, effect sizes are likely to be small. 150 patients were recruited to the study and they were all followed up for a period of three months from the time of admission, as risk factors persist after discharge home.

It must be remembered that focusing on a specific problem such as the occurrence of DVT, to evaluate the predictive accuracy of the DVT scale, can lead to a source of error. In this study, the applications of preventative measures were not excluded because of legal and ethical considerations.

The efficacy of mechanical and pharmacological prophylaxis has been so firmly confirmed (Kakkar 1990, Kakkar et al, 1997) that it would be considered a serious omission of "duty of care" and clinical negligence not to administer prophylaxis to the needy. 81 out of the 150 patients (54%) received some form of known and proven prophylaxis, and this could have influenced the DVT outcome and explain the poorer sensitivity of the DVT scale.

If the results of nursing studies are to be applied to nursing practice, then the nursing community needs assurance that the effects are substantial enough and supported by statistical evidence. Future studies on the ongoing evaluation of the DVT scale must involve more patients, so as to obtain greater effect size and power. The most obvious and straightforward method is to increase the sample size.

The versatility of the DVT scale was demonstrated in its application across the three clinical directorates without having to modify its actual design. Some items were easier to test in one area than the other. The trauma risk category predominantly applies to the orthopaedic patients and the high risk disease category to the medical patients as is the surgical intervention category to the patients on the general surgical ward. Its application and evaluation in diverse clinical specialities will allow for the testability of all the items and provide further evidence to its portability and universal application. A single cutoff score range of 15 $\geq$ applied across the medical, orthopaedic and surgical patients yielded different levels of sensitivity, specificity, positive predictive and negative predictive values due to the nature of the specialities (Table 6.2). A ROC plot must be applied by users of the DVT scale to optimise its predictive accuracy as one cutoff score value may not be appropriate for all.

Table 6.2: Sensitivity, specificity, positive and negative predictive values of DVT scale across the three specialities

Specialities	Cutoff scores	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Orthopaedics N= 50	15≥	25	83	36	90
Medical N= 50	15≥	8	95	33	77
Surgical N=48	15≥	25	98	67	87

Noting that sensitivity and specificity are inversely related, a low sensitivity of the DVT scale is punctuated by a very high specificity and a low predictive value for cases is significantly outweighed by a high predictive value for negatives.

A comparative analysis of the Autar DVT scale

The DVT scale has demonstrated several advantages over other forms of risk assessment strategies. A broad-brush guesstimate of "potential problem of DVT" as used on pre-printed care plan (Autar, 1994) provides only a very crude index of risk assessment. Unless risk is quantified and patients classified into risk categories, clinical decision making in terms of what constitutes the best prophylaxis, is likely to be ineffective (THRiFT, 1998; International Consensus Statement, 1997; 2001).

On the other hand, the revalidated Autar DVT risk assessment protocol (figure 7:p187) uses quantifiable clinical data to rank patients into one of the three levels of DVT risk. This strategy makes the whole DVT assessment process visible and guides practitioners into implementing the most appropriate venous thromboprophylaxis as recommended by the national and international consensus groups.

It is in the area of primary prevention of DVT that the Autar DVT scale has exhibited considerable promises. Essentially, there are two types of venous thromboprophylaxes: primary and secondary. Primary prophylaxis is about preventing DVT. DVT is most preventable and appropriate venous prophylaxis reduces its occurrence by 50% (Vanek, 1991). On the other contrary, secondary prevention is directed at treating DVT once it occurs, in order to prevent extension of the condition or fatal complications such as PE

or PPS. Risk factors are additive and the crux of primary prophylaxis is to quantify risk in patients, so that the recommended intervention can be promptly initiated. Calculating DVT risk objectively in an individual within a multifactoral aetiology, the DVT scale allows for this problem to be addressed proactively. This proactive approach to DVT management is in very sharp contrast to the reactive nature of the problem, whereby DVT is treated, as it occurs to prevent extension its immediate serious complications. Wells et al (1994) develop a clinical prediction guide to confirm any provisional suspected DVT, so that it can be promptly and safely treated. This reactive approach to DVT is very costly to the health of the individual and socio-economically.

Consistency and practical application are other two areas where the Autar DVT scale outperforms alternative strategy such as haematological data for detecting hypercoaguability.
Although several researchers (Clayton et al, 1976: Crandon et al, 1980 and Lowe et al, 1982) have used the same predictive index and on similar population, the outcomes have very dissimilar and inconsistent. This is because the laboratory procedure lacks standardisation, yielding different cutoff scores and values (Palareti et al, 1991) and are forbiddingly complex to interpret (Gallus,1987). On the other hand, the high percentage agreement, kappa values and intra-class correlation coefficients of the DVT scale are proof to the consistency of its application.
Another problem with laboratory testing is that it can only be initiated by medical staff at the exclusion of nurses and other health care professionals who play a key role in the prevention of DVT. Additionally, this heavy dependency on laboratory data to predict risk, means that venous thromboprophylaxis cannot be implemented, until rheological test results are available. On the other hand, the Autar DVT scale relies on routine data gathered on admission, allowing for a prompt DVT risk assessment and timely interventions. Like the universally recognised Glasgow coma scale, the Autar DVT scale can be applied by nurses and doctors alike and other health care professionals

The use of computer programmed with logistic regression formulas, as devised by Janssen (1987) is technology for the future. Until artificial

intelligence is available for carrying bedside assessment, paper and pencil assessment tools as the DVT scale, remain the most cost-effective method of predicting risk and guiding decision making. The DVT risk factors are readily assessable items and day to day observations undertaken by nurses. This confers the advantage of generating a pragmatic DVT risk assessment instrument. It takes only a few minutes to complete, requiring only minimal education and training and no other formal measurements or additional equipments.

Summary

The Autar DVT scale has demonstrated good consistency in five reliability studies. However, it is also recognised that the existing reliability of the instrument can be increased by the simple clarification of several variables. The term "acute myocardial infarction" is preferred to explain an acute episode and to rule out "myocardial infarction" as a past medical event. Staff must be reminded constantly to choose either from the trauma risk category of the subscale or the surgical intervention category, as not to overstate risk. The layout of the revalidated scale is designed to minimise this kind of human error. Clarifying orthopaedic surgery as below waist procedures (figure 7) in the surgical intervention category, differentiates such high risk procedures from other types of orthopaedic surgery carrying less risk of DVT.

In terms of its predictive validity, the DVT scale correctly classified 78 per cent of the patients. The inverse relationship between specificity and sensitivity meant a high specificity of 90 per cent was achieved against a low sensitivity of 25 per cent. A high predictive value of 84 per cent of negative outcome was accomplished at a cost of a predictive value of 37 per cent of positive outcome. Refining some of the covariates of the tool could improve its predictive validity. As DVT increases exponentially with advancing age, and the patients over the age of 70 is set to rise by almost a half, a fifth element is included in the age specific category subscale. Patients in the 70+ group are assigned a high risk score of 5.

Evidence is now overwhelmingly supportive of the association between HRT and DVT. HRT is an addition to the special risk category and is assigned a

risk score of 2. Hereditary Thrombotic Disease is increasingly recognised as a high risk factor and Thrombophilia is another addition to the special risk category with a risk score of 4.

On the other hand, despite recent publicity, air travel as risk factor is inconclusive and not considered in the DVT scale. Haematological conditions such as haemolytic anaemia and sickle cell anaemia, previously linked with DVT, have been removed from the high risk disease subscale, due to lack of supporting evidence. Opinion is divided on varicose veins as an independent variable. Current evidence acknowledges varicose veins as an associated factor but concedes the high risk is not what it is made out to be. Varicose veins risk is assigned a revised risk score of 3. The covariate CVA has a revised score of 6 to differentiate it from the slightly higher risk posed by a previous DVT.

In order to reduce the rate of false positives, the risk assessment protocol has been reduced to three risk categories, namely low, moderate and high risk. This classification is consistent with the recommended risk assessment protocol of the international consensus group. Overall, the DVT scale has exhibited good practical application and can be applied to guide clinical decision making, in relation to the most appropriate prophylaxis. Finally, future studies on the ongoing evaluation of the DVT scale must involve more patients, so as to obtain greater effect sizes and power

References

ACCP (1996) Associated College of Chest Physicians Consensus Committee on Pulmonary Embolism. Opinions regarding the diagnosis and management of venous thromboembolic disease. Chest .109: 233-237.

Agu O (1999) Deep Vein thrombosis: A risky business. Further correspondence. British Journal of Theatre Nursing. Vol 9, No 7: 290-291.

Ahmad Z & Mohyuddin Z (1996) Deep vein thrombosis as a predictor of cancer. Angiology. Vol 47, No 3: 261-265.

Allotey JC, Louca O (1997) Thromboembolic Disorders: treatment and diagnosis. British Journal of Midwifery. Vol 5, No 2: 75-79.

Altman DG, Bland MJ (1994) Diagnostic test 2: predictive values. British Medical Journal.Vol 309: 102.

Anands SS, Wells PS, Hunt D et al (1998) Does the patient have deep vein thrombosis? JAMA. Vol 279 No 14: 1094-1099.

Anderson FA, Wheeler HB, Goldberg RJ et al (1991) A population-based perspective of the hospital incidence and case fatality rates of Deep Vein Thrombosis and Pulmonary Embolism: the Worcester DVT study. Arch Intern Med. 151: 933-938.

Anderson FA, Wheeler HB, Goldberg RJ et al (1994) Changing Clinical Practice. Acta Intern Med. Vol 154: 669-673.

Anderson FA, Wheeler HB (1995) Venous Thromboembolism: Risk Factors and Prophylaxis. Clinics in Chest Medicine. Volume 16, Number 2: 235-251.

Anthony D (1999) Understanding Advanced Statistics. A Guide for Nurses and Health Care Researchers, Churchill Livingstone, Edinburgh.

Antman EM, Lau J, Kupelnick B et al (1992) A comparison of results of meta-analyses of randomized controlled trials and recommendations of clinical experts: treatments for myocardial infarction. JAMA 268: 240-248.

Arcelus JI, Traverso CI, Caprini JA (1991) Venous Thromboembolism Prophylaxis and Risk Assessment in Medical Patients. Seminars in Thrombosis and Hemostasis, Vol 17, Supplement 3: 313- 318.

Armentrout G (1993) A comparison of the medical model and the wellness model the importance of knowing the difference. Holistic Nursing Practice. Vol 7, No 4: 57-62.

Ashby EC, Ashford NS, Campbell MJ (1995) Posture, blood velocity in common femoral vein and prophylaxis of venous thromboembolism. Lancet 345: 419-421.

Autar R (1994) Nursing Assessment of Clients at risk of DVT: the Autar DVT scale.
Unpublished MSc dissertation. University of Central England. Birmingham, England.

Autar R (1996a) Nursing assessment of clients at risk of deep vein thrombosis (DVT): the Autar DVT scale. Journal of Advanced Nursing.23: 763-770.

Autar R (1996b) The Scope of Professional Practice in Specialist Practice. British Journal of Specialist Nursing. Vol 5,No 16: 984-990.

Autar R (1996c) Deep Vein Thrombosis: the silent killer. Quay Books. Mark Allen Publishing.Wiltshire.

Autar R (1998) Calculating patients' risk of deep vein thrombosis. British Journal of Nursing. Vol 7, No 1: 7-12.

Autar R (1999) Deep Vein Thrombosis: a risky business. British Journal of Theatre Nursing. Vol 9 No 3: 114-115.

Badaracco MA, Vessey MP (1974) Recurrence of venous thromboembolic diseases and the use of oral contraception. British Medical Journal 1: 215-217.

Bahal V, Silverman SH (1993) The case for stratification analysis when prescribing deep vein thrombosis prophylaxis. J. R Coll. Surg. Edin. 38: 33-35.

Banga JD, Sixma JJ (1986) Diabetes Mellitus, Vascular Disease and Thrombosis. Clinics in Haematology. Vol15, No2: 465-487.

Barnes RW (1982) Current Status of Non Invasive Tests in the Diagnosis of Venous Diseases. Surgical Clinics of North America.Vol 62,No 3: 484-500.

Becker DM (1986) Venous Thromboembolism: Epidemiology, Diagnosis, Prevention. Journal of General Internal Medicine. Vol 1: 402- 411.

Beisher NA, Mackay EV (1986) Normal Puerperium. Anatomy and Physiology. Normal care. Obstetrics and the Newborn. An illustration textbook.second edition, Bailliere Tindall, London.pp 519-523.

Belcaro G, Nicolaides AN, Veller M (1995) Venous disorders. A Manual of Diagnosis and Treatment. WB Saunders Company Ltd. London.

Belcher AE (1993) Blood Disorders. Mosby's Clinical Nursing Sciences. Philadelphia.

Bell WR, Simon TL, De Mets DL (1977) The clinical features of submissive and massive pulmonary emboli. American Journal of Medicine. 62: 355.

Bell WR, Simon TL (1982) Current status of pulmonary thromboembolic disease: Pathophysiology, diagnosis, prevention, and treatment. Curriculum in Cardiology.American Heart Journal: 239- 262.

Benner P (1984) From Novice to Expert. Addison Wesley, Menlo Park, California.

Bennett B, Towler HM (1985) Haemostatic response to trauma. Br Med Bulletin. 41:274-280.

Beral V (1977) Mortality among oral contraceptive users. The Lancet. Vol. 2: 727-731.

Bergqvist D, Lindblad B (1985) A 30 year survey of pulmonary embolism verified at autopsy: an analysis of 1274 surgical patients. British Journal of Surgery. 72: 105-108.

Bergqvist D (1988) Concluding remarks and remaining problems on thromboprophylaxis. Acta Chir Scand. Suppl 543: 48-51.

Bernstein CN, Blanchard JF, Houston DS et al (2001)The incidence of Deep Venous Thrombosis and Pulmonary Embolism among patients with Inflammatory Bowel Disease. A population-based cohort study. Thrombosis Hemostasis. 85: 430-434.

Bevan L (2000) West Country Success. Practice Nurse. Vol. 19, No 5: 208-211.

Bland J M (1995) An introduction to Medical Statistics. 2nd edition, Oxford Medical Publication.

Bland MJ & Altman DG (1996) Measurement error and correlation coefficients. British Medical Journal. Vol 313: 41-42.

Bland J M, Altman DG (2000) The odds ratio. British Medical Journal 320,1468

Bonnar, McNicol GP, Douglas AS (1969) Fibrinolytic enzyme system and pregnancy. British Medical Journal.111: 387-389.

Bonnar J (1981) Venous Thromboembolism in Pregnancy. Clin Obstret Gynacol. 8: 155-173.

Bonnar J (1987) Coagulation effects of oral contraception. American Journal of Obstetrics and Gynaecology. Vol. 157,No 4(2) 1042-1048.

Borrow M, Goldson H (1981) Post-operative venous thrombosis: evaluation of five methods of treatment. Am J Surg.141: 245-251.

Blenner JL (1993) Development of the Stimulus Intensity Modulation Scale. Journal of Nursing Measurement. 1: 5-18.
Boston Collaborative Drug Surveillance Program (1974) Surgically confirmed gallbladder disease, venous thromboembolism and breast tumour

in relation to post menopausal oestrogen therapy. New England Journal of Medicine. 290: 15-19.0

Boulay F, Berthier F, Schoukroun G et al (2001) Seasonal variations in hospital admission for deep vein thrombosis and pulmonary embolism: analysis of discharge data. British Medical Journal. Vol 323: 601-602.

Bournemeax H, Hicklin L, Desmarais S (1996) Seasonal variations in deep vein thrombosis. British Medical Journal. Vol312: 284-285.

Bowling A (1995) Measuring disease. Open University Press. Buckingham.

Brach B, Moser K, Cedar L (1977) Venous thrombosis in acute spinal paralysis. J Trauma. 17: 289-293.

Brandjes Dees PN, Buller H, Heijhoer H et al (1997) Randomised trial of effects of compression stockings in patients with symptomatic proximal- vein thrombosis. Lancet. Vol 349: 759-762.

Brennan PF, Hays BJ (1992) The kappa statistic for establishing interrater reliability in the secondary analysis of quantitative clinical data. Research in Nursing and Health. 15: 153-158.

Bret A, Ferree MD, Stern PJ et al (1993) Deep Venous thrombosis after spinal surgery. Spine. Vol. 18,No3: 315-319.

British National Formulary (1994) BNF: A joint publication of the BMA and the Royal Pharmaceutical Society of Great Britain. No 27: 301-308.

British National Formulary (2000) BNF: a joint publication of the BMA and the British Pharmaceutical Society of Great Britain. No 39: 368-369.

Broekmans AW, Bertine RM, Loeliger EA (1983) Protein C and the development of skin necrosis during anticoagulant therapy. Thromb Haemostat. 49: 244.

Brown DC, Neumann RD (1995) Orthopaedic Secrets.Hanley, Belfus Inc/ Philadelphia.

Brown R, Taylor FC, Cohen H (1998) Setting up a nurse-led anticoagulant clinic. Professional Nurse. Vol14, No 1: 21-23.

Browse NL, Gray L, Morland M (1977) Changes in the blood fibrinolytic activity after surgery. Br J Surgery. 64: 23-27.

Brunner L, Suddarth D (1992) The Textbook of Adult Nursing. Chapman and Hall, London.

Byrne GT, McCarthy MJ, Silverman S (1996) Improving uptake of prophylaxis for venous thromboembolism in general surgical patients using prospective audit. British Medical Journal. Vol313:917.

Byrt T, Bishop T, Carlin JB (1993) Bias, Prevalence and kappa. Journal of Clinical Epidemiology. 46: 423-429.

Callum M (1992) Prevalence of Chronic Leg Ulceration and Severe Chronic Venous Disease in Western Countries. Phlebology. Suppl 1: 6-12.

Campbell J (1993) Making sense of blood groups. Nursing Times. Vol 89, No 22: 36-38.

Campbell MJ, Machin D (1993) Medical Statistics. A common sense approach. Second edition. John Wiley & Sons Ltd. Chichester.pp 60-68.

Campbell MJ (2001) Statistics At Square Two. BMJ books. London.

Campbell WB, Ridler BMF (1995) Varicose vein surgery and deep vein thrombosis. British Journal of Surgery. 82:1494-1497.

Campbell B (1996) Thrombosis, plebitis and varicose veins. British Medical Journal. Vol 312: 198-199.

Capper C (1999) External Pneumatic Compression Therapy for DVT Prophylaxis. British Journal of Theatre Nursing. Vol 9, No3: 109-110.

Caprini JA, Scurr J& Hasty JH (1988) Role of Compression Modalities in a Prophylactic Program for Deep Vein Thrombosis. Seminars in Thrombosis and Hemostasis.(Supplement) Vol 14: 77-87.

Caprini JA, Natonson RA (1989) Postoperative Deep vein Thrombosis: Current Clinical Considerations. Seminars in Thrombosis and Hemostasis. Vol. 15,No 3: 244-249.

Caprini JA, Arcelus, JI Hasty JH et al (1991) Clinical Assessment of Venous Thromboembolic Risk in Surgical Patients. Seminars in Thrombosis and Hemostasis. (Supplement 3), Vol 17: 304-312.

Caprini JA, Arcelus JI, Hoffman BA et al (1994) Prevention of Venous Thromboembolism in North America. Results of a survey among general surgeons. Journal of Vascular Surgery. Vol 20,No5: 751-757.

Carroll P (1993) Deep venous Thrombosis: Implication for Orthopaedic Nursing. Orthopaedic Nursing. Vol 12,No 3:33-43.

Carter CJ, Gent M, Leclerc JR (1987) The epidemiology of venous thrombosis. In: Colman RW, Hirsh J, Marder VJ, Salzman EW (eds) Hemostasis and thrombosis: basic principles and clinical practice. Second edition. JB Lippincott Company, Philadelphia.

Carruthers M, Arguelles AE, Mosovich A (1976) Man in transit: biochemical and physiological changes during intercontinental flights. Lancet.1: 977-980.

Carter CJ (1992) Thrombosis in relation to oral contraceptives and hormone replacement therapy. In: Haemostasis and Thrombosis in Obstetrics and

Gynaecology. (Eds Greer IA, Turpie AGG,Forbes CD) London, Chapman and Hall: 371-385.

Castledine G (1996) Nurses must not become the mechanical hands of doctors. British Journal of Nursing. Vol 5, No 6: 386.

Castledine G (1997) Evidence Based Nursing: Where is the evidence? British Journal of Nursing. Vol 6,No 5: 290.

Castledine G (1997) The abuse of specialist nursing titles must stop. British Journal of Nursing. Vol 6, No 19: 1137.

Chakrabarti R, Hocking ED, Fearnly GR (1969) Reaction of three stresses-Electroplexy-Surgery and Myocardial Infarction. - of Fibrinolysis and Plasma Fibrinogen. Journal of Clinical Pathology. Vol 22: 659-662.

Christopoulos D, Nicolaides AN, Cook A et al (1989) Pathogenesis of venous ulceration in relation to the calf muscle pump function. Surgery. 829-835.

Claggett GP, Reisch JS (1988) Prevention of Venous Thromboembolism in General Surgical Patients. Annals of Surgery. Vol 208,No 2: 227-240.

Clagett GP, Anderson FA, Levine MN, Salzman E, Wheeler HB (1992) Prevention of Venous Thromboembolism. Chest. Vol 102, No 4: 391S – 407S.

Clagett GP, Anderson FA, Heit J ey al (1995) Prevention of Venous Thromboembolism. Fourth ACCP Consensus Conference on Antithrombotic Therapy. Chest. 108 : 312S – 334S.

Clayton JK, Anderson JA, McNicol GP (1976) Preoperative prediction of postoperative deep vein thrombosis. Br Med Journal. 2: 910-912.

Cohen JA (1960) A coefficient of agreement for nominal scales. Educational and Psychological Bulletin. 20: 37-46.

Cohen JA (1968) Weighted kappa: nominal scale with provision for scales disagreement or partial credit. Psychological Bulletin. Vol 70, No. 4:213-220.

Cohen J (1977) Statistical power analysis for the behavioural sciences. Academic Press, New York.

Cohen JA (1988) Statistical power analysis for behavioural sciences (2nd edition) Hillsdale. NJ Erlbaum.

Coleridge-Smith JP, HastyJH, Scurr J H (1990) Venous stasis and vein lumen changes during surgery. Br J Surgery. Vol 77:1055-1059.

Collins R, Scrimgeour A, Yusuf S et al (1988) Reduction in fatal pulmonary embolism and venous thrombosis by perioperative administration of subcutaneous heparin. New England Journal of Medicine 318: 1162-1173.

Comerata AJ, Stewart GJ, Alburger BD et al (1989) Operative venodilation: a previously unsuspected factor in the cause of post-operative deep vein thrombosis. Surgery. Vol 106, No 2: 301-309.

Comp PC (1986) Hereditary disorders predisposing to thrombosis. Prog Hemostasis Thromb. 8: 71.

Connoughton M (2001) Evidence based coronary care. Churchill Livingstone, Edingburgh.pp 40.

Cook MK, Sanders S, Bagshaw M, Finnie RM (2001) A woman is anxious about Deep Venous Thrombosis on a long haul flight. Coronary Health Care. Vol 5, No 2: 100-104.

Cooligan H (1996) Introduction to Research Methods and Statistics in Psychology. 2nd edition. Hodder & Stoughton,London.

Coon WW, Willis (1973) Recurrence of venous thromboembolism. Surgery.Vol 73 No 6: 823-827.

Coon WW, Willis PW, Keller JB (1973) Venous thromboembolism and other venous disease in the Tecumseh community health study. Circulation. 48: 839.

Coon WW (1976) Epidemiology of venous thromboembolism. Ann Surg 186(2) 149-164.

Coon WW (1977) Risk factors in Pulmonary Embolism. Surgery, Gynaecology and Obstetrics.143: 385-390.

Coon WW (1984) Venous thromboembolism: Prevalence, risk factors and prevention. Clinics in Chest medicine. Vol 5, No 3:391- 401.

Cooper DN (1994) The molecular genetics of familial venous thrombosis. Bailliere's Clinical Haematology. Vol 7,No 3: 637-674.

Cormack DFS, Reynolds W (1992) Criteria for evaluating the clinical and practical utility of models used by nurses. Journal of Advanced Nursing. Vol 17: 1472-1478.

Cotton LT, Roberts VC (1977) The prevention of deep venous thrombosis with particular reference to mechanical methods of prevention. Surgery. 81: 228-237.

Crandon AJ, Peel KR, Anderson JA, Thompson V, McNicol GP (1980) Post-operative deep vein thrombosis: identifying high risk patients. Br Med Journal. 7: 343- 344.

Crookes P, Davies S (1998) Research into Practice. Bailliere Tindall. London.

Crow J (1977) The Nursing Process -2. How and why to take a nursing history. Nursing Times. 73: 950- 957.

Cruickshank JM, Gorlin, Jennett B (1988) Air travel and thrombotic episodes: the economy class syndrome. Lancet. Vol. 2: 497-498.

Dalen JE, Paraskos JA, Ockene IS et al (1986) Venous thromboembolism. Scope of the problem. Chest. Suppl 371S-373S.

Daly E, Vessey MP, Painter R et al (1996) Risk of venous thromboembolism in users of hormone replacement therapy. Lancet. 348: 977-980.

da Vaus DA (1993) Surveys in social research. London, University College of London.

Das SK (1994) Venous Thrombosis and Pulmonary Embolism. Surgery. 217-223.

Davis P (1998) The hidden threat: deep vein thrombosis. Journal of Orthopaedic Nursing. 2: 45-51.

Deagle J (1998) Hospital at home: Deep vein thrombosis (Low molecular weight heparin anticoagulant therapy). Primary Health Care. Vol 8, No 9: 23-25.

Deeks J (1996) Pressure sore prevention: using and evaluating risk assessment tools. British Journal of Nursing. 5(5): 313-319.

Deykin D (1970) Warfarin therapy. New England Journal of Medicine. 283: 801-803.

Dismuke SE, Wagner EH (1986) Pulmonary Embolism as a cause of death. JAMA. Vol 255,No 15: 2039-2042.

DoH (1982) Report on Confidential Enquiries into Maternal deaths in England and Wales. 1976-1978. DHSS 26: HMSO, London.

DoH (1993) Vision for the future. HMSO, London.

DoH (1994) Report on Confidential Enquiries into Maternal Deaths in the United Kingdom, 1988-1990. HMSO, London.

DoH (1996) Report on the Confidential Enquiry into Maternal Deaths. 1991-1993. HMSO, London.

DoH (1977) The New NHS. Modern. Dependable.HMSO, London.

DoH (1998) Our healthier Nation.HMSO, London.

DoH (2001) National service framework for older people. HMSO, London.

Doll R, Peto R (1976) Mortality in relation to smoking: 20 years' observation in male British doctors. British Medical Journal. 2: 1525- 1536.

Douglas A (1978) Venous Thrombosis and Pulmonary Embolism: a disease of the hospitals. Nursing Mirror. Vol 147: 44-46.

Dowling S, Martin R, Skidmore P et al (1996) Nurses taking on junior doctor's work: a confusion of accountability. British Medical Journal. 312: 1211-1214.

Drug Therapeutics Bulletin (1992) Preventing and treating Deep Vein Thrombosis. Vol 30, No 3: 9-12.

Edwards FC, Truelove SC (1964) The course and prognosis of Ulcerative Colitis. Gut. 5: 1-15.

Eklof B, Kistner RL, Masuda EM et al (1996) Venous Thromboembolism in association with prolonged air travel. Dermatologic Surgery. Vol 22, No 7:637-641.

emedicine.com,Inc (2001) Deep Venous Thrombosis and Thrombophlebitis: 1-24.

Emerson PA, Marks P (1977) Preventing thromboembolism after Myocardial Infarction: effect of low dose heparin or smoking. Br Med Journal. 1:18-20.

Erd felder E, Faul F, Buchner A (1996) G Power: a general power analysis programme. Behaviour Research Methods, Instruments and computer. 28: 1-11. Heinrich Heine University. Dussuldorf.

Ernst E, Hammerschmidt D, Bagge U et al (1987) Leukocytes and the risk of ischemic disease. JAMA. 257: 2318-2324.

Essex-Sorlie D (1995) Medical Biostatics and Epidemiology. First edition. Prentice Hall International Inc, London.

European Consensus Statement (1991) Prevention of venous thromboembolism. Med-Orion Publishing, London.

Faculty of Family Planning and Reproductive Health care (1995) Response for doctors to the Committee on Safety of Medicine's letter of 18th October 1995. Family Planning Association. London.

Fegan C (1998) Tinzaparin as an antithrombotic: an overview. Hospital Medicine. Vol 59,No 2: 145-148.

Feinstein AR, Cicchetti DV (1990) High Agreement but Low kappa: 1. The problems of two paradoxes. Journal of Clinical Epidemiology. Vol 42, No 6: 543-549.

Ferrari E, Chevallier T, Chapelier A, Bandouy M (1999) Travel as a risk factor for venous thromboembolic disease: a case control study. Chest. 115: 440-444.

Flanc C, Kakkar VV, Clarke MD (1969) Post-operative deep vein thrombosis: effect of intensive prophylaxis. Lancet.1: 477.

Fleiss JL (1971) Measuring nominal scale agreement among many raters. Psychology Bulletin.Vol 76, No 5: 378.

Fleiss JL (1981) Statistical methods for rates and proportions. 2nd ed. New York. John Wiley and sons.

Fletcher RH, Fletcher S, Wagner Edward H (1996) Clinical epidemiology: the essentials. 3rd edition, Williams and Wilkins. Baltimore.

Fowler JF (1993) Survey Research Methods. 2nd edition. Sage Publications, London.

Freeark RJ, Boswich J, Fardin R (1967) Post traumatic venous thrombosis. Arch Surg. 95: 567-575.

Friedel H, Balfour J (1994) Tinzaparin. A review of its pharmacology and clinical potential in the prevention and treatment of thromboembolic disorders. Drugs. 48: 638-660.

Frisbie JH, Sasahara AA (1981) Low dose heparin prophylaxis for deep vein thrombosis in acute spinal cord injury patients: a controlled study. Paraplegia. 19: 343-346.

Gallerani M, Manfredini R, Ricci L et al (1992) Sudden death from pulmonary embolism: chronobiological aspects. European Heart Journal. 13: 661-665.

Gallus AS (1976) Venous thromboembolism incidence and clinical risk factors. In: Madden JL, Hume M, and (eds). Appleton-Century Crofts: 1-32. New York

Gallus AS, Hirsh J, O'Brien SE et al (1976) Prevention of venous thrombosis with small subcutaneous doses of heparin. JAMA. 235:1980-1982.

Gallus AS (1989) Overview of Management of Thrombosis and Hemostasis. Vol 15, No 2: 99-109.

Gallus AS, Salzman EW, Hirsh J (1994) Prevention of Venous Thromboembolism. In: Colman RW, Hirsh J, Marder VJ, Salzman EW, (eds) Homeostasis and Thrombosis: Basic Principles and Clinical Practice. Third edition. JB Lippincott Company, Philadelphia.

Geerts WH, Code KI, Jay RM et al (1994) A prospective study of venous thromboembolism after major trauma. The New England Journal of Medicine. Vol 331,No 24: 1601-1606.

Geroulakos G (2001) The risk of venous thromboembolism for air travel: the evidence is only circumstantial. British Medical Journal. Vol. 322: 188.

Gibbon B (1998) Validity and reliability of assessment tools. Nurse researcher. Vol 2,No 4: 48-55.

Gibbon B (1998) Selecting healthcare assessment tools: putting issues of validity and reliability into a wider context. Nurse Researcher. Vol. 5 No 3: 5-15.

Gibbs NM (1957) Venous thrombosis of the lower limbs with particular reference to bed-rest. Br J Surg. Vol XIV. No 191: 15-236.

Goldberg RJ, Seneff H, Gore JM et al (1987) Occult malignant neoplasm in patients with deep venous thrombosis. Arch Intern Med. 147: 251-253.

Goldhaber SZ, Savage DD, Garrison RJ (1983) Risk Factors for Pulmonary Embolism. The Framingham Study. The American Journal of Medicine. Vol. 74: 1023-1028.

Gordis L (1996) Epidemiology. WB Saunders Company. Philadelphia.

Gorman WP, Davis KR, Donelly R (2000) Swollen Lower Limbs-1: general assessment and deep vein thrombosis: In ABC of Arterial and Venous Diseases. Eds Donelly R & London N, 55-59. BMJ Books.

Gosnell DJ (1993) An assessment tool to identify pressure sores. Nursing Research. 22: 55-59.

Goucke CR (1989) Prophylaxis against venous thromboembolism. Aesth Intens Care 17(4) 458- 465.

Gows S, MacGilluray I (1971) Metabolic, hormonal and vascular changes after synthetic oestrogen therapy in oophrectomized women. British Medical Journal. 2:73.

Grace R (1993) Thromboprophylaxis: a review. Br J Hos Med. 49 (10) 720-726.

Graf V, Baggenstosis A, Sauer WG et al (1966) Venous Thrombosis occurring in non specific ulcerative colitis. A necropsy study. Archive of Internal Medicine. Vol 117: 377-382.

Gray M (1997) Evidence based Healthcare. How to make Health Policy and Management Decisions. Edinburgh. Churchill Livingstone.

Green SM (1997) Obesity, prevalence, causes, health risk and treatment. British Journal of Nursing. Vol 6,No 20: 1181-1185.

Griffin JH, Evatt B, Zimmerman TS (1981) Deficiency of Protein C in congenital thrombotic disease. J Clin Invest. 68: 1370-1373.

Grimshaw JM, Russel IT (1993) Effects of clinical guidelines on medical practice: a systemic review of rigorous evaluations. The Lancet. Vol 343: 1317-1322.

Grodstein F, Stampfer MJ, Goldhaber SZ et al (1996) Prospective study of exogenous hormones and risk of pulmonary embolism in women. Lancet. 348: 983-987.

Guilleband J (1985) Surgery and the Pill. British Medical Journal. 291: 498-499.

Hamilton HW, Cranford JS, Gardiner JH et al (1970) Venous thrombosis in patients with fracture of the upper end of the femur. J Bone Joint Surgery. 52: 268-289.

Handley AJ, Tether D (1974) Influence of smoking on deep vein thrombosis after Myocardial Infarction. British Medical Journal. 3:230-231.

Harkness GA (1995) Epidemiology in Nursing Practice. Mosby Year book. St Louis, Missouri.

Hartmann D (1977) Consideration in the choice of interobserver reliability estimates. Journal of Applied Behavior Analysis. 10: 103-116.

Hass S (1997) Managing the risk of venous thromboembolism in orthopaedics: concluding remarks. Orthopedics. (Supppl): 26-27.

Havig O (1977) Deep Venous Thrombosis and Pulmonary Embolism. Arch Chir Scand. Supplement 478: 48-80.

Health Service Circular (1999) Clinical Governance: Quality in New NHS. NHS Executive, London.

Hefley WF, Nelson CL, Pushkarich-May CL (1996) Effect of delayed admission to the hospital on the preoperative prevalence of deep vein thrombosis associated with fractures about the hip. Journal of Bone and Joint Surgery. 78-A (4) 581-583.

Herzog JA (1992) Deep Vein Thrombosis in the Rehabilitation Clients. Rehabilitation Nursing. Vol 17,No 4: 196-198.

Hirsh J (1977) Hypercoagulability. Seminar in Hematology. 14: 409-425.

Hirsh J, Genton E, Hull R (1981) Venous thromboembolism. Grune & Stratton, New York.

Hirsh J (1981) Blood tests for the diagnosis of venous and arterial thrombosis. Blood. 57:1-8.

Hirsh J, Hull R (1982) Natural history and clinical features of venous thrombosis. In: Colman RW, Hirsh J, Marder V, Salzman EW (eds) Hemostasis and Thrombosis. JB Lippincott, Philadelphia.pp 834.

Hirsh J, Dalen JE, Deykin D,Poller L (1992) Oral anticoagulants. Mechanism of action, clinical effectiveness, and optimal therapeutic range. Chest. 102:312.

Homans J (1954) Thrombosis of the deep leg veins due to prolonged sitting. The New England Journal of Medicine. Vol 250,No4: 148-149.

House A, Farber J, Nier L (1983) Differences in computational accuracy and speed of calculation between three measures of interobserver agreement. Child Study.13: 195-201.

Howell RW (1970) Smoking habits and laboratory tests. Lancet. 2: 152.

Huber O, Bournemeaux H, Borst et al (1992) Post operative pulmonary embolism after hospital discharge: an underestimated risk. Archive of Surgery. Vol 127:310-313.

Hull R, Hirsh J, Sackett DL et al (1981) Clinical validity of a negative venogram in patients with clinically suspected venous thrombosis. Circulation. 64: 622-625.

Hull RD, Hirsh J, Sacket DL, Stoddart G (1981) Cost effectiveness of clinical diagnosis, venography and non invasive testing in patients with symptomatic deep vein thrombosis. The New Journal of Medicine. Vol. 304,No 26: 1561-1567.

Hull R, Raskob GE, Gent M et al (1990) Effectiveness of Intermittent Pneumatic Leg Compression for preventing Deep Vein Thrombosis after Total Hip Replacement. JAMA. Vol. 263,No 17: 2313-2317.

Hull RD (1992) Venous Thromboembolism in Spinal Patients. Chest. Vol102, No 6:Supplement 658S- 663S.

Hunt BJ, Jurd KM (1998) Endothelial Cell Activation: a central pathophysiological process. British Medical Journal. Vol 316: 1328-1329.

Ingram R (1998) Power analysis and sample size estimation.NT research. Vol 3, No 2:132-140.

International Consensus Statement (1997) Prevention of venous thromboembolism. Med-Orion Publishing Company, London.

International Consensus Statement (2001) Prevention of venous thromboembolism. International Angiology, Vol 20,No 1: 1-37.

Jebb S (2000) A weighty issue. Nursing Times. Vol 96, No 36: 32-33.

Jick H, Slone D, Westerholme B (1969) Venous thromboembolic disease and ABO blood type. Lancet. 1: 539.

Jick H, Derby IE, Myers MW (1996) Risk of hospital admission for idiopathic venous thromboembolism among users of post menopausal oestrogens. Lancet. 248: 981-983.

Joffe S (1975) Incidence of postoperative deep vein thrombosis in neurosurgical patients. Journal of Neurosurgery. Vol 42: 201-203.

Jones DRB, McIntyre IMC (1975) Venous thromboembolism in infancy and childhood, Arch Dis Child. 50: 153.

Jordan K, Ong BN, Croft P (1998) Mastering Statistics. A Guide for Health Service Professionals and Researchers. Stanley Thornes Ltd. Cheltenham.

Jowett N I, Thompson DR (1989) Comprehensive Coronary Care. Scutari Press, London.

Kaisary A (1982) Leg ulcer. Aetiology and pathogenesis of venous leg ulcer. Nursing. (2nd series) suppl: 1-7.

Kakkar V V, Howe C, Nicolaides AN, Renney JTG, Clark MB (1970) Deep Vein Thrombosis of the legs. Is there a high risk group? Am J Surg. 120:527-530.

Kakkar VV, Lawrence D (1985) Haemodynamic and clinical assessment after therapy for acute deep vein thrombosis. The American Journal of Surgery. Vol 150:No 4:54-63.

Kakkar VV (1990) Prevention of Venous Thrombosis and Pulmonary Embolism. The American Journal of Cardiology. Vol. 65: 51C-54C.

Kakkar VV, Stringer MD (1990) Prophylaxis of venous thromboembolism. World Journal of Surgery.14: 670-678.

Kakkar VV, Boeckl), Boneu B et al (1997) Efficacy and safety of low molecular weight heparin and standard unfractionated heparin for prophylaxis of post operative venous thromboembolism. World Journal of Surgery. 21: 2-9.

Kamal A (1987) Cerebro-Vascular Disease and its management. Wolfe Medical Publication LTD, London.

Kendall (1992) The guide to protocol development for the prevention of deep vein thrombosis and pulmonary embolism. Kendall Health Care Products, Europe.

Kerlinger FN (1986) Foundations of behavioural research (3rd ed). New York: Holt, Rinehart and Winston.

Kesteven PLJ (2000) Traveller's thrombosis. Thorax. 55 (suppl 1) S32-S36.

Kierkegaard A (1983) Incidence and diagnosis of Deep vein thrombosis associated with pregnancy. Acta Obtetrica et Gynecologica, Scandinavica. 62: 239-243.

Kierkegaard A, Lars N, Olson CG, Castenfor J et al (1987) Incidence of DVT in bedridden non surgical patients.Acta Med Scand. 222: 409-414.

Knapp TR (1996) The overemphasis on power analysis. Nursing Research. Vol 45, No 6: 379-380.

Koran LM (1975) The Reliability of Clinical Methods, Data and Judgements. The New England Journal of Medicine. Part 1, Vol. 293, No 4: 642-646.

Kudsk KA, Fabian TC, Baum S et al (1989) Silent Deep Vein Thrombosis in Immobilised Multiple Trauma Patients. The American Journal of Surgery. Vol 158:515-518.

Lam A, Border IT, Inwood MJ et al (1975) Coagulation studies in Ulcerative Colitis and Crohn's disease. Gastroenterology. 68:245-251.

Lambie JM, Mahaffy RG, Barber DC (1970) Diagnostic accuracy in venous thrombosis. British Medical Journal. Vol 2: 142.

Landis JR, Koch GC (1977) The measurement of observer agreement for categorical data. Biometrics. 33: 159.

Lang WE (1983) Glucocorticoids inhibit Plasminogen Activator production by endothelial cells. Thrombosis Hemostasis. 50: 888-892.

Larsen E (1986) Evaluating Validity of Screening Tests. Nursing Research. Vol 35, No: 186-188.

Lassen MR, Borris LC (1991) Mobilisation after hip surgery and efficacy of thromboprophylaxis. Lancet. 337 :618.

Letsky E (1985) Coagulation Problems during Pregnancy. 29-45. Churchill Livingstone, Edinburgh.

Letsky EA (1989) Coagulation defects: In De Swiet M, ed. Medical Disorders in Obstetric Practice. 2nd edition, Blackwell Scientific, Oxford. 104-165.

Levine M, Gent M, Hirsh J et al (1996) A comparison of Low Molecular Heparin administered primarily at home with Unfractionated Heparin administered in the hospital for proximal Deep Vein thrombosis. The New England Journal of Medicine. Vol 334,No 11: 677-681.

Lewin L, Wakefield J (1979) Percentage agreement and phi. A conversion table. Journal of Applied behavior Analysis. 12: 299-301.

Lilienfield AM, Lilienfield DE (1980) Foundation of Epidemiology. 2nd ed. Oxford University Press. pp150-159.

Lindblad B (1988) Prophylaxis of postoperative thromboembolism with low dose heparin alone or in combination with dihydroergotamine. A review. Acta chir Scand. (Suppl) 31-42.

Lindblad B, Sternby NH, Bergqvist D (1991) Incidence of venous thromboembolism verified by necropsy over 30 years. BMJ. Vol 302:709-711.

Lindsay GM, Gaw A (1997) Coronary Heart Disease Prevention. A handbook for the health care team. Churchill Livingstone. Edingburgh.

Lowe GD, McArdle BM, Carter DC et al (1982) Prediction and selective prophylaxis of venous thrombosis in elective gastro-intestinal surgery. Lancet.1: 409-412.

Lowe GD, Lee AJ, Rumley A et al (1992) Epidemiology of haematocrit, white cell count, red cell aggregation and fibrinogen. Clinical Hemorheology.12: 535-544.

Lowe GD (1993) Laboratory investigation of pre-thrombotic states: In Poller L, Thomson JM (eds) Thrombosis and its management. Churchill Livingstone, Edingburgh. pp31-45.

Lowe GD, Woodward M, Vessey MP et al (2000) Thrombotic variables and risk of idiopathic venous thromboembolism in women aged 45-64 years. Relationships to hormonal replacement therapy. Thrombosis and Haemostasis. 83(4) 530-535.

Lowe L (1979) The role of anticoagulants in hip surgery. In: McKibbin B, ed. Recent Advances in Orthopaedics 3. Edinburgh, Churchill Livingstone.

Lyne PA, Papanikolaou P, Lycette E (2000) An empirical investigation of pressure ulcer risk factors. Nursing Standard.14, 49: 46-53.

Macmurdo B 91991) Risk watch: safety critical products. Occupational Safety and Health. Vol 21, No 10: 9-10.

Maclure M, Willett M (1987) Misinterpretation and misuse of the kappa statistics. American Journal of Epidemiology. Vol 126, No. 2: 161-169.

Mahoney I, Barthel DW (1965) Functional Evaluation in the Barthel Index. Maryland State Medical Journal. Vol 14:61-65

Maling JJB (1998) Oral Contraceptives and venous thromboembolism-managing the uncertainty. Adverse Drug Reaction Bulletin. Lippincott, Williams & Wilkin. Philadelphia.

Mammen EF (1992) Pathogenesis of Venous Thrombosis. Chest. Vol 102 No 6: 640s-644s.

Mansfield AO (1972) Alteration in fibrinolysis associated with surgery and venous thrombosis. British Journal of Surgery. Vol 59, No14: 754-757.

Marks P, Emerson PA (1974) Increased incidence of deep vein thrombosis after Myocardial Infarction in non smokers. B Med Journal. 3: 232-234.

Marlar RA, Mastovitch S (1990) Hereditary Protein C deficiency. A review of the genetics, clinical presentation, diagnosis and treatment. Blood Coagulation Fibrinolysis. 1: 319-330.

Maurer BJ, Wray R, Shillingford JP (1971) Frequency of venous thrombosis after myocardial infarction. The Lancet. 1386-1387.

Marsh N 91981) Fibrinolysis. John Wiley & Sons. Chichester.10-45.

Mausner JS, Kramer S (1985) Epidemiology. An introductory Text. WB Saunders Co, Philadelphia.

May R: (1979) Varicose Veins: In May R (ed): Surgery of Veins of the leg and Pelvis, Stuttgart, Thieme,

McCarthy,ST, Robertson D, Turner JJ, Hawkey CJ (1977) Low dose heparin as a prophylaxis after acute stroke. The Lancet. 800-801.

Meade TW (1983) The thromboembolic disorders: the epidemiology of atheroma and thrombosis. FK Schattauer Verlag, New York. 116-125.

Mehrens WA, Lehmann IJ (1978) Measurement and evaluation in education and psychology. 2nd edition. Holt,Rinehart and Winston, New York.

Merli GJ, Martinez J (1987) Prophylaxis for deep vein thrombosis and pulmonary embolism in surgical patients. Medical Clinics of North America. 71: 377-397.

Merli GJ, Herbison GJ, Ditunno JF et al (1988) Deep Vein thrombosis: Prophylaxis in Acute Spinal Cord Injured Patients. Arch Physical Med Rehabil. Vol 69: 661-664.

Millbank (1970) The epidemiology of venous thrombosis. The Millbank Memorial Fund Quarterly. Whole issue.

Miller RC, Lies JE, Caretta RF et al (1976) Prevention of lower extremity venous thrombosis by early mobilisation. Annal of Internal Medcine. 84: 700.

Mills C (1997) Pulmonary embolism. Nursing Times. 93 (51): 50-53.

Milne R (1992) Venous thromboembolism and travel: is there an association? Journal of the Royal College of Physicians of London. Vol 26: No 1: 47-49.

Moore B (1976) Sequential Mestranol and Novethisterone in the treatment of climateric syndrome. Postgraduate Medical Journal. 52(6) 39-47.

Mol W, EgbertsTCG (1994) Prophylaxis for venous thromboembolism in hip fracture surgery: total costs and cost effectiveness in Netherlands. PharmacoEconomics. 5, No 1: 48-55.

Monterey JS, Kistner RC, Kong AYT et al 91983) Thromboembolism after hip fracture. Journal of Trauma. Vol 85: No 25: 534-537.

Morrell MT, Dunnill MS (1968) The post mortem incidence of pulmonary embolism in a hospital population. British Journal of surgery. 55: 347-352.

Morris AS (1985) Screening in Chronic Disease. New York, Oxford University Press

Morris E (1998) HRT: Still a role in CVD. Geriatric Medicine. Vol 28, No 8: 27-31.

Mourante AE, Kopec AC, Kazimiera DS (1971) Blood Groups and Blood Clotting. Lancet. 1:223.

Moser KM (1989) Pulmonary Embolism: your challenge is prevention. The Journal of Respiratory Diseases. Vol 10: No 10: 83-91.

Moser KM, (1994) Diagnosing pulmonary embolism. British Medical journal. 309: 1525-1526.

Moyses C (1988) Economy class syndrome. Lancet. Vol 2: 1077.

Munro BH (2001) Statistical Methods for Health Care Research. Lippincott, Philadelphia.

Myllenen P, Kammomen M, Rokhanen P et al (1985) Deep venous Thrombosis and Pulmonary Embolism in Patients with Acute Spinal Cord Injury: a comparison with non paralysed patients immobilised due to Spinal fractures. J Trauma. 25: 541-543.

National Association of Health Authorities and Trusts (NAHAT 1991) National briefing: medical negligence. Birmingham. No 14: 1-5.

NHS Executive (1996) Clinical guidelines. HMSO, London.

National Institutes of Health (1986) Consensus development conference on the prevention of venous thrombosis and pulmonary embolism. J Am Med Ass. 25b: 744-749.

NCEPOD (1993) Report of the National Confidential Enquiry into Peri Operative Deaths (1991/1992). HMSO. London.

Nelzen O, Bergqvist D, Lindhagen A (1991b) Leg Ulcer etiology. A cross sectional population study. Journal of Vascular Surgery. 14: 557-564

Nelzen O, Bergqvist D, Lindhagen A et al (1991b) Chronic leg ulcers: an underestimated problem in the primary health care among elderly patients. J Epidemiol Comm Health. 45: 184-187.

Nicolaides AN, Kakkar VV, Renny JJG et al (1971) Myocardial infarction and DVT. British Medical Journal. 1: 432-434.

Nicolaides AN, Irving D (1975) Clinical Factors and the Risk of Deep Venous Thrombosis: In Nicolaides AN ed, Thromboembolism Aetiology. Advances in prevention and management. MTP, Lancaster:pp 193-204.

Nicolaides AN (1990) Benefits of prophylaxis in general surgery. Acta Chir Scand. Suppl 556: 25-29.

Nilsson PE, Bergqvist D, Benoni G et al (1997) The post discharge prophylactic management of the orthopedic patients with Low Molecular Weight Heparin: Exoparin.Orthopedics. Feb suppl: 26-27.

Norbeck JS (1985) What constitutes a publishable report of instrument development? Nursing Research. Vol. 34,No 6: 380-382.

Nordstrom M, Linblad B, Berqvist D, Kjellstrom (1992) A prospective study of the incidence of deep vein thrombosis within a defined urban population. Journal of Internal Medicine. 232: 155- 160.

Nordstrom M, Lindblad B, Anderson H et al (1994) Deep venous thrombosis and occult malignancy: an epidemiological study. British Med Journal. Vol. 38: 891-894.

Norrell ES (1995) Workbook of Epidemiology. Oxford University press.

Norton D, McClaren R, Exton-Smith AN (1962) An investigation of geriatric nursing problems in hospital. London. National Corporation for the care of old people.

Notelovitz M, Ware M (1982) Coagulation risk with post menopausal oestrogen therapy. In: Progress in Obstetrics and Gynaecology (ed Studd JWW). Churchill Livingstone, Edingburgh. 2: 228-240.

Nursing and Midwifery council (2002) Code of professional conduct. April 2002. London.

Nylander G, Semb H (1972) Veins of the lower limb after tibial fractures. Surg Gyn /Obs. 134 :974-976.

Office for National Statistics (1995) Mortality Statistics DH2 (22). Government Statistical service, London.

Ogston D (1987) Venous Thrombosis: causation and prevention. John Wiley & Sons Ltd. Chichester.

Office of Health Economics (1995) Compendium of Health Statistics. OHE, White Crescent Press Ltd. Luton.

OHE (1996) Office of Home Economics. Deep Vein Thrombosis and Pulmonary Embolism. White Crescent Press Ltd, Luton.

Oppenheim AN (1992) Questionnaire design, Interviewing and Attitude Measurement. Pinter Publishers, London.

OPCS (1990) Mortality Statistics Cause: England and Wales. A Publication of the Government Statistical Service. DH2, No 17. HMSO. London.

Oster G, Tuden,RL, Colditz GA (1987) A cost effectiveness analysis of prophylaxis against deep vein thrombosis in major orthopaedic surgery. JAMA. Vol 257, No 2: 203- 208.

Paiement GD, Mendelsohn (1997) Risk of Venous Thromboembolism in Orthopedic patient: Epidemiological and Physiological Data. Orthopaedics. Supplement:7- 9.

Palareti G, Maccafer M, Manotti C et al (1991) Fibrinogen assays: a collaborative study of six different methods. Clinical Chemistry. 37: 714-719.

Parahoo K (1997) Nursing research. Principles, Process and Issues. Macmillan Press. London.

Parker-Williams J, Vickers R (1991) Major orthopaedic surgery on the leg and thromboembolism. Br Med J. 303: 531-532.

Pearson TC, Wetherby-Mein G (1978) Vascular occlusive episodes and venous haematocrit in primary proliferative polycythaemia. Lancet. 2: 1219.

Perez- Gutthann S, Rodriguez G, Castellsagne J et al (1997) Hormone replacement and risk of venous thromboembolosm. British Medical Journal. 314: 796-800.

Perkash A, Prakash V, Perkash I (1978) Experience with the management of thromboembolism in patients with spinal cord injury. Part 1: Incidence, diagnosis and role of some risk factors. Paraplegia. 16: 322-331.

Perkins JMT, Galland BB (1999) Venous thrombosis and pulmonary Embolism: Part 1. Prevention. Care of the critically ill. Vol. 15,No 4:140-142.

Petaja J, Myllynen P, Rokhanen P et al (1989) Fibrinolysis and Spinal Injury: relationship to post traumatic deep vein thrombosis. Acta Chir Scand. 155: 241-246.

Pineo GF, Brann MC, Gallus AS et al (1976) Tumors, mucus production and hypercoagulability. Ann NY Acad Sci. 230: 262-270.

Polit DE, Hungler B (1985) Essentials of Nursing Research. JB Lippincott Company, Philadelphia.

Polit DE, Sherman RE (1990) Statistical Power in Nursing Research. Nursing Research. Vol 39, No 6: 365-369.

Polit DE (1996) Data analysis and statistics for Nursing Research. Appleton & Lange. Connecticut.

Polit DE & Hungler B (1999) Nursing Research. Principles and Methods. 6th Edition. Lippincott. Philadelphia.

Poller L (1978) Oral contraceptives, Blood Clotting and Thrombosis. British Medical Bulletin. Vol 347, No 2: 151-156.

Poller L, Taberner DA (1982) Dosage and control of oral anticoagulants: an international survey. British Journal of Haematology. 51:479.

Poller L (1993) Recent Advances in Blood Coagulation. 6. Churchill Livinsgstone, Edinburgh.

Pout G, Winperis J, Dilks G (1999) Nurse-led outpatient treatment of deep vein thrombosis. Nursing Standard. 13 (19) 39-41.

Prandoni P, Mannucci PM (1994) Deep Vein thrombosis of the lower limbs: diagnosis and management. Bailliere's Clinical Haematology. Vol 7,No 3: 693- 701.

Prandoni P, Anthonie WA, Lensing MD et al (1996) The long term clinical course of acute deep vein thrombosis. Annals of Internal Medicine. Vol 125,No 1: 1-7.

Prescott RJ, Jones DRB, Vasilescu C et al (1978) Smoking and Risk factors in Deep Vein Thrombosis. Thrombosis Haemostat. (Stuttg) 40:128- 133.

Priest J, McColl E, Thomas RN et al (1995) developing and refining a new measurement tool. Nurse Researcher. Vol 2,No 4: 69-81.

Rakoczi I, Chamone D ,Cohen D , Verstraete M (1978) Prediction of postoperative leg vein thrombosis in gynaecological patients. Lancet.1:509-510.

RCOG (1995) Report of the Royal college of Obstetricians and Gynaecologists Working party on Prophylaxis Against Thromboembolism in Gynaecology and Obstetrics. Chameleon Press Limited. London.

Redman HC, (1988) Deep Venous Thrombosis: Is contrast venography still the diagnostic " Gold Standard"? Radiology. 168: 277-278.

Reid E (1999) Pulmonary embolism: an overview of treatment and nursing issues. British Journal of Nursing. Vol 8, No 20: 1373-1378.

Reynolds W, Cormacks DFS (1991) An evaluation of the Johnson Behavioural System Model. Journal of Advanced Nursing. 16: 1122-1130.

Rickles FR, Edwards RL (1983) Activation of blood coagulation in cancer: Trousseau 's syndrome revisited. Blood. Vol 62,No1:14-31.

Roberts GH (1963) Venous Thrombosis in hospital patients: a postmortem study. Scottish Medical Journal. 8: 11-15.

Roberts TS, Nelson CL, Barnes L et al (1990) the preoperative prevalence and postoperative incidence of thromboembolism in patients with hip fractures treated with dextran prophylaxis. Clinical Orthopaedics and Related Research. 255: 198-203.

Rocha E, Alfaro MJ, Paramo JA, Canadell JM (1988) Pre-operative identification of patients at high risk of deep vein thrombosis despite prophylaxis in total hip replacement. Thrombosis Haemostasis. 59: 93-95.

Rolling PS (1995) Deep Venous thrombosis: In Brown DE and Newman RD, Orthopaedic Secrets.pp 30-38, Hanley and Belfus, inc/ Philadelphia.

Rose SD (1979) Prophylaxis of Thromboembolic Disease. Medical Clinics of North America. Vol 63, No 6: 1205-1224.

Rosendaal FR (1997) Thrombosis in the Young: Epidemiology and Risk factors. A focus on Venous Thrombosis. Thrombosis and Haemostasis. Vol 78, No 1: 1-6.

Rosendaal FR (1999) Venous Thrombosis: a multi causal disease. The Lancet. Vol 353: 1167-1173.

Rossi EC, Green D, Rosen JS et al (1980) Sequential Changes in Factor VIII and Platelets Preceding Deep Vein Thrombosis in Patients with Spinal Cord Injury. Br J Haematol. 45: 143-151.

Royal College of General Practitioner Oral contraceptive study (1978) Oral Contraceptives, Venous Thrombosis and Varicose Veins. Journal of the Royal College of General Practitioner. 28: 393.

Ruckley CV (1985) Protection against thromboembolism. Br J Surg. 72 (6) 421-422.

Sackett D, Haynes RB, Guyatt GH, Tugwell P (1991) Clinical Epidemiology. A Basic Science for Clinical Medicine. Second Edition. Little, Brown and Company. Toronto

Sackett D, Rosenberg WMC, Muir Gray JA et al (1996) Evidence based medicine: What is and what it isn't? British Medical Journal. Vol312: 71-72.

Salzman EW & Hirsh J (1982) Prevention of venous thromboembolism: basic principles and clinical practice: In Coleman RW, Hirsh J, Salzman EW (eds). Hemostasis and Thrombosis. Lippincott, New York. P 982.

Salzman EW (1986) Venous Thrombosis made easy. The New England Journal of Medicine. Vol 314,No 13: 847-848.

Salzman EW & Hirsh J (1987) Prevention of venous thrombosis: In Coleman RW, Hirsh J, Marder VJ, Salzman EW (eds). Hemostasis and Thrombosis. Philadelphia,Pa: Jb Lippincott: 1252-1265.

Samama MM, Simmoneau G, Wainstein JP (1993) Sirius study. Epidemiology of risk factors of deep venous thrombosis (DVT) of the lower limbs in community practice. Thrombosis Haemostasis. Vol 69: 763.

Sandler DA, Martin JF (1989) Autopsy proven pulmonary embolism in hospital patients: Are we detecting enough deep vein thrombosis? Journal of Royal Society of Medicine. 82: 203-205.

Sartwell PE, Masi AT, Arthes FG (1969) Thromboembolism and oral contraceptives: an epidemiological case control study. American Journal of Epidemiology. 90: 365.

Sartwell PE, Stolley PD (1982) Oral contraceptives and Vascular Disease. Epidemiological Review. 4: 95-109.

Schaub N, Duckert F, Fridrick R, Gruber UF (1975) Post-operative venous thrombosis. Lagenbeck Arch Chir .340: 23.

Scurr JH, Coleridge-Smith PD, Hasty JH (1987) Regimen for improved effectiveness of intermittent pneumatic compression in deep venous thrombosis prophylaxis. Surgery. Vol. 102, No 5: 816-820.

Scurr JB, Coleridge-Smith PD, Hasty JB (1988) Deep Vein thrombosis: a continuing problem. B Med J. 297: 28.

Scurr JH (1990) How long after surgery does risk of thromboembolism persists? Acta Chir Scand. Suppl 556: 22-24.

Serjeant GR (1992) Sickle Cell Disease.2nd edition. Oxford University Press. Oxford. pp 117-119.

Sevitt S & Gallagher (1961) Venous thrombosis and Pulmonary embolism: a clinico-pathological study injured and burned patients. Br J Surg. 48:475-498.

Sevitt S & Gallagher (1969) Prevention of venous thrombosis and pulmonary embolism in injured patients. Lancet. 2: 981-989.

Shackford SR, Moser KM (1989) Deep Venous Thrombosis and Pulmonary Embolism in Trauma Patients.Journal of Intensive Care Medicine. Vol 3, No 2:87-98.

Shafer AJ (1985) The hypercoagulable states. Ann Intern Med. 102:814.

Sharnoff JG, Rosenberg M (1969) Effects of age and immobilisation on the incidence of post operative thromboembolism. Lancet. 1: 845.

Sharnoff JG (1980) Prevention of venous thrombosis and pulmonary embolism. Lancaster. MTP Press.

Sigel B, Ipsen J, Felix WR (1974) The epidemiology of lower extremity deep venous thrombosis in surgical patients. Annals of Surgery. 179: 278-290.

SIGN (1995) Scottish Intercollegiate Guidelines Network. Prophylaxis of venous thromboembolism. A national clinical guideline recommended for use in Scotland.

Silver JR, Morris WR, Ostfinkskowski JS (1980) Associated injuries in patients with spinal injury. Injury. 12: 219-224.

Simmons AV, Shepperd MA, Cox AF (1973) Deep venous thrombosis after MI. British Heart Journal. 35: 623-625.

Simms KA, Fought SG (1989) Clinical decision making in critical care. Critical Care Nursing Quarterly. Vol 12, No 3: 79-84.

Singer A (1983) Bed rest: deep vein thrombosis and pulmonary embolism. (Letter) JAMA. 250, No 23: 3162.

Sioson ER, Crowe WE, Daeson NV (1988) Occult proximal deep vein thrombosis: its prevalence among patients admitted to a rehabilitation hospital. Arch Phys Med Rehab. Vol 69: 183-185.

Soeken KL, Prescott PA (1986) Issues in the use of kappa to estimate reliability. Medical Care.Vol 24:733-741.

Sorenson RM, Pace NL (1992) Aesthetic Techniques during Surgical repair of femoral Neck Fractures. Anaesthesiology. 77: 1095-1104.

Sproul EE (1938) Carcinoma and venous thrombosis: the frequency of association in the body or tail of the pancreas with multiple venous thrombosis. American Journal of Cancer.34: 566-585.

SPSS 11.0 (2001) Application Guide. SPSS Inc, Chicago.

Stadel BV (1981) Oral contraceptives and cardiovascular disease. New England journal of Medicine. Vol 305: 612-618.

Stamatakis JD, Sagar S, Naird D (1977) Femoral vein thrombosis and total hip replacement. BMJ. 2:223-225.

Stewart-Rose J (1998) Deep vein Thrombosis: the risk. Journal of Orthopaedic Nursing. 2: 130-131.

Stolley PD, Tonascia JA, Tuckman MS et al (1975) Thrombosis with low estrogen oral contraceptives. American Journal of Epidemiology. 102: 197-208.

Stoppard M (2001) Why hasn't HRT given me relief? The Mirror. (15th August): 27.

Strandness DE, Langlois Y, Cramer M et al (1983) Long term sequelae of acute venous thrombosis. JAMA. 146: 1289-1292.

Sue-Ling HM, Johnson D, Mc Mahon MJ, Philips PR (1986) Pre-operative identification of patients at risk of deep vein thrombosis after elective major abdominal surgery.Lancet. 1: 1173-1176.

Sue-Ling H, Hughes LE (1988) Should the pill be stopped preoperatively. British Medical Journal. 296: 447-448.

Swann KW, Black PM (1984) Deep vein thrombosis in Neurosurgical Patients: a review. Journal of Neurosurgery. 61:1055-1062.

Symington IS & Stack BHR (1977) Pulmonary thromboembolism after travel. Chest. 71: 138-140.

Tabachnick BG & Fidell LS (1996) Using Multivariates Statistics. 3rd Edition,Harper Collins College Publishers. New York.

Talbot RW, HeppelJ, Dozois R et al (1986) Vascular complications of inflammatory bowel disease. Mayo Clinic. Vol 61: 140-145.

Talbot LA (1995) Principles and Practice of Nursing Research. Mosby. A Times Mirror Company. Baltimore.

Tardy B, Page Y, Zeni Fet al (1993) Phlebitis following travel. Presse Medicale. 22, No 17: 811-814.

Taylor F, Gray A, Cohen H et al (1999) Evaluation of a nurse specialist anticoagulant service. Clinical Laboratory and Haematology. 19: 267-272.

Teasdale AM (1975) Assessing conscious level. Nursing Times.71 (24): 914-917.

THRiFT (1992) Risk of and Prophylaxis for Venous Thromboembolism in Hospital Patients. BMJ.Vol 305: 567-574.

THRiFT (1998) Risk of and Prophylaxis for Venous Thromboembolism in Hospital Patients. Phlebology. 13: 87-97.

Tomalin DA, Redfern SJ, Norman IJ (1992) Monitor or Senior Monitor: problem of administration and some proposed data. Journal of Advanced Nursing. 17(1): 72-82.

Tomalin DA, Oliver S, Redfern SJ & Norman IJ (1993) Inter-rater reliability of Monitor, Senior Monitor and Qualpacs. Journal of Advanced Nursing. 18: 1152-1158.

Topf M (1986) Three estimates of interrater reliability for nominal data. Nursing Research.35 (4): 253-255.

Trousseau A (1865) Phlegmasia alba dolens. Clinique Medicale de L'Hotel-Dieu de Paris, London. New Sydenham Society. 3:94.

Truelove SC (1984) Ulcerative Colitis. Update Postgraduate Centre Series. Update Publications Ltd, London.

Turpie AGG, Hirsh J, Jay RM et al (1987) Double- blind randomised trial of Org 10172 low molecular weight heparinoid in prevention of deep vein thrombosis in thrombotic stroke. The Lancet. 523-526.

Turpie AGG (1997) Low Molecular weight Heparin: From the bench to the orthopaedic patients. Orthopedics. February supplement: 10-13.

Tuthill V (1995) The training of nurse surgical assistants. Surgical Nurse. 4 (21) 1240-1245.

UKCC (1992) The Scope of Professional Practice. UKCC, London.

UKCC (1994) The future of professional practice: the Council's standards for education and practice following registration. UKCC, London.

UKCC (1999) A higher level of practice. Report of the consultation on UKCC's proposals for a revised regulatory framework for post-registration clinical practice. UKCC, London.

Valette F, Hoffmeyer V, Llyod A (1995) Economic evaluation of the use of Tinzaparin in the treatment of deep vein thrombosis. Br J Med Econ. 8: 111-123.

Vandenbroucke JP, Van der Meer FJM, Helmerhorst FM et al (1996) Factor V Leiden: Should we screen oral contraceptive users and pregnant women? British Medical Journal. Vol 313:1127-1130.

Vanek VW, Gantt N, Spirto G (1991) Grand Rounds. Review of the literature and recommendations for prophylaxis of Deep Vein thrombosis and Pulmonary Embolism in surgical and trauma patients. Current Surgery. 539-553.

Van De Pette J, Mackie C (1996) Clinics led by nurse practitioners can work well. British Medical Journal. Vol 312: 1226.

Ventura MR, Hageman PT,Slakter MJ et al (1980) Interrater reliabilities for two measures of nursing care quality. Research in Nursing and Health. 3: 25-32.

Verstraete M (1997) Prophylaxis of venous thromboembolism. British Medical Journal. Vol 314: 123-125.

Vessey MP, Doll R (1969) Investigation of the relation between use of oral contraceptives and thromboembolic disease. A further report. B Medical Journal. 3: 651-657.

Vessey MP, Doll R (1970) Post operative thromboembolism and the use of oral contraceptives. British Medical Journal.3: 123-126.

Virchow R 1846) Uber die Verfpfung der Lungenarterie. Nue Notisen und Geb D Natur Heilk. Vol 36: 26.

Wallis M, Autar R (2001) Deep Vein Thrombosis: clinical nursing management. Nursing Standard. 15,No 18: 47-54.

Waltz CF, Strickland OL, Lenz ER (1991) Measurement in Nursing Research. Philadelphia. FA Davis Company.

Warlow C, Ogston D, Douglas AS (1972) Venous thrombosis following strokes. Lancet. 1: 1305-1306.

Warlow C, Ogston D, Douglas AS (1976) Deep venous thrombosis of legs after stroke. Part 1- Incidence and predisposing factors. British Medical Journal. 1: 1178-1181.

Warlow C (1978) Venous thromboembolism after stroke. Am Heart J, 96 (3): 283-285.

Warkentin TE, Levine MN, Hirsh J et al (1995) Heparin Induced Thrombocytopenia in patients treated with low molecular weight heparin and unfractionated heparin. The New England Journal of Medicine. Vol 332, No 20: 1330-1335.

Washing CC, Moss H (1988) Pragmatic Aspects of Establishing Interrater Reliability in Research. Nursing Research. Vol 37, No 3: 190-191.

Wassermann LR, Goldberg JDBalcerzak SP et al (1981) for the Polycythaemia Study group: influence of therapy on cause of death in polycythaemia Vera. Clinical Research. 29:573A.

Wasson JH, Sox HC, Neff RK et al (1985) Clinical prediction rules. The New England Journal of Medicine. Vol 313, No 13: 793-799.

Waterlow J (1985) A risk assessment card. Nursing Times. 81(49): 51-55.

Watson N (1968) Venous Thrombosis and Pulmonary Embolism in Spinal Cord Injury. Paraplegia. 6:113-121.

Webb C (1989) Action Research: philosophy, methods and personal experience. Journal of Advanced Nursing. 14: 403-410.

Weinmann EE, Salzman EW (1994) Medical Progress: Deep Vein Thrombosis. The New Journal of Medicine. Vol 331, No 24: 1630-1641.

Wells PS, Anderson DR, Kearon C et al (1993) The combination of pretest clinical likelihood (PCL) and non invasive tests improves management of outpatients suspected with deep vein thrombosis (DVT). Thrombosis & Haemostasis. 69: 1080.

Wells P, Lensing AW, Hirsh J (1994) Efficacy of Graduated Compression Stockings: a meta analysis. Arch Intern Med. Vol. 154: 62-72.

Wells PS, Hirsh J, Anderson D et al (1995a) Accuracy of clinical assessment of deep vein thrombosis. Lancet. 345: 1326-1330.

Wells PS, Brill-Edwards, Stevens P et al (1995b) A Novel and rapid whole-blood assay for D-Dimer in patients with clinically suspected Deep Vein Thrombosis. Circulation. 91:2184 - 2187.

Wheeler HB (1988) Venous Thromboembolism following trauma. Journal of Intensive Care Medicine. 3: 65-66.

Wheeler HB, Hirsh J, Wells P et al (1994) Diagnostic Tests for Deep vein Thrombosis. Arch Intern Med Vol 154: 1921-1928.

White A, Nicolass G,Foster K et al (1993) Health surveys for England 1991. HMSO, London.

Wille-Jorgensen P, Thorup J, Fisher A et al (1985) Heparin with and without graded compression stockings in the prevention of thromboembolic complications of major abdominal surgery: a randomised trial. Br Journal of Surgery. 72: 579-581.

Willmers J, Bergqvist D, Hallbrook T (1982) Venous insufficiency as a late complication of tibial fracture. Acta Orthop Scand. 53: 149-153.

Wilmshurst P (1994) Temperature and cardiovascular mortality. British Medical Journal. 309: 1029-1030.

Wood ME, Bunn PA (1994) Hematology/ Oncology Secrets. Harley, Belfus Inc/ Philadelphia.

Wolfe W, Sarbiston D (1980) Pulmonary Embolism. Saunders, Philadelphia.

World Health Organisation (1995) Venous Thromboembolic disease and combined oral contraceptives: results of international multicentre case control study. Lancet. Vol. 346: 1575-1582.

Wright HP, Osborn SB, Edmons DG (1951) Effects of postoperative bed rest and early ambulation on the rate of venous blood flow. Lancet. 1:22-25.

Wyshock E, Caldwell M, Crowley JP 91988) Deep Venous thrombosis, inflammatory bowel disease and protein S deficiency. American Journal of Intensive Care Medicine. 3: 633-635.

Yarandi HN, Simpson SH 91991) The Logistic Regression model and the Odds of testing HIV positive. Nursing Research. Vol 40, No 6: 372-373

Yelnik A, Dizien O, Bussel B et al (1991) Systemic Lower Limb Phlebography in Acute Spinal Cord Injury in 147 patients. Paraplegia. 29:253-260.

Zahn HR, Skinner JA, Porteous MJ (1999) The preoperative prevalence of deep vein thrombosis in patients with femoral neck fractures and delayed operation. Injury. Vol 30: 605-607.

Zweig MH, Campbell G (1993) Receiver Operating Characteristic (ROC) Plots: A Fundamental Evaluation Tool in Clinical Medicine. Clinical Chemistry. Vol 39, No 4: 561-577.

Advancing clinical practice in the management of Deep Vein thrombosis (DVT). Development, application and evaluation of the Autar DVT risk assessment scale.

Volume 2 of 2

Appendices

Thrombosis Risk Factor Assessment
Medical and Surgical Patients

Due within 24 hours of admission.

Name _____ Age _____ Sex _____

Diagnosis _____ Admission: Elective or Emergency

Type of surgery planned _____

Please check all pertinent factors (Each risk factor has value of one unless otherwise noted).

- ☐ Age 41 to 60 years
- ☐ Age 61 to 70 years (2 factors)
- ☐ Age over 70 years (3 factors)
- ☐ Anticipated bed confinement over 72 hours
- ☐ History of DVT/PE (3 factors)
- ☐ Varicose veins
- ☐ Obesity (>20% of ideal body weight)
- ☐ History of previous major surgery
- ☐ Previous immobilisation (>72 hours)
- ☐ MI
- ☐ CHF
- ☐ Stroke
- ☐ Crystalloid infusion (>5 litres/24 hours)
- ☐ Severe COPD
- ☐ Trauma
- ☐ Planned operation over 2 hours
- ☐ Pelvic surgery or total joint replacement
- ☐ Confining travel, air/rail/road (>4 hours within week of admission)
- ☐ History of pelvic or long bone fracture
- ☐ Leg oedema, ulcers, stasis
- ☐ Malignancy
- ☐ Pregnancy or post-partum (<1 month)
- ☐ Inflammatory bowel disease
- ☐ Severe infection
- ☐ Hormone therapy
  - Name _____
  - Dosage _____
- ☐ Hypercoagulable states
  - Congenital _____
  - Acquired _____
- ☐ Other _____

☐ LOW RISK (*1 factor*)	☐ MODERATE RISK (*2–4 factors*)	☐ HIGH RISK (*>4 factors*)
IN-PATIENT T.E.D. Thigh Length Stockings + early ambulation	IN-PATIENT T.E.D. Thigh Length Stockings + SCD/Heparin*	IN-PATIENT T.E.D. Thigh Length Stockings + SCD/Heparin/Warfarin*
	Regimen:	Regimen:
DISCHARGE ResT.E.D. Below Knee Stockings + early ambulation	DISCHARGE ResT.E.D. Thigh Length Stockings + early ambulation	DISCHARGE ResT.E.D. Thigh Length Stockings + early ambulation

*Delete as appropriate

Other instructions _____

C/I to anticoagulants ☐ Yes ☐ No

If yes, explain _____

_____

Prescriber's signature _____ Date _____

Arcelus J I et al, *Venous Thromboembolism Prophylaxis and Risk Assessment in Medical Patients*
Seminars in Thrombosis and Hemostasis, Vol 17, 3, 1991

Caprini J A et al, *Clinical Assessment of Venous Thromboembolic Risk in Surgical Patients*
Seminars in Thrombosis and Hemostasis, Vol 17, 3, 1991

LEICESTERSHIRE HEALTH AUTHORITY (Appendix 2)

Project Id:

Date Received:

Decision Code:

Committee on the Ethics of Clinical Research Investigation
Application for Research Ethics Approval

The following application form is for submission to the Ethics Committee for approval of proposed medical research involving human subjects*. Please ***complete the form in typescript*** and return to the address given at the bottom of this page for approval.

The following must also be submitted with the protocol:

Detailed Protocol
Questionnaires Used
Proposed Consent Form
Patient Information Leaflet
CTX (if appropriate)
Confirmation of compensation arrangements

N.B. The Ethics Committee in considering an application for ethical approval bears in mind the Royal College of Physicians guideline that ***"badly planned, poorly designed research that causes inconvenience to subjects and may carry risk without producing useful or valid results, is unethical."***

Notes for completing application

Please complete every section of the form as fully as possible. The shaded areas are only for office use, so please do not fill these in. ***Respond to all Yes/No questions by circling the appropriate answer.***

Address: Director of Public Health
Ethics Committee
Leicestershire Health
Gwendolen Road
Leicester, LE5 4QF

If your work will be done in the LRI, LGH or GGH, please send this form to the Research and Development Office of your Trust, who will forward it to the address above.

1. Title Of Project

Advancing clinical practice in the management of Deep Vein Thrombosis(DVT).

Development, application and evaluation of the Autar DVT Scale.

2. Miscellaneous Details

2.1 Where will the research be done? (tick **one** as appropriate)

LRI	/
LGH	
GGH	/
Other hospital	
GP	/
Other location	

If other please state where: Community Hospitals-

2.2 Starting Date (DD-MON-YY): AS AP

2.3 Duration (in months): 48

3. Responsible Investigator (Supervisor of Project)

N.B. This is the individual with overall responsibility of the proposed study, not necessarily the individual who will be carrying out the study.

3.1 Name: STUART BRAND Title(Dr):

Address:School of Health&Policy Studies
University of Central England
Perry Barr
Birmingham
B42 2SU.

Position/post held (Consultant, Senior Nurse etc.): Principal Lecturer

Qualification:BSc(Hons)PHYSIOLOGY(Manchester University)
PhD(PHYSIOLOGY)Leicester University)

3.2 List the individual(s) who will be carrying out the study.

Name	Position/Post Held	Qualification
A R AUTAR	SENIOR LECTURER	BA(Hons)MSc,RN,RMN, DN(Lond)ENB 100,CERT ED,RNT
Nurses from negotiated wards	Registered Nurses	Pre-registration & Registered Nurses

Person to whom correspondence should be addressed:

A R AUTAR
Senior Lecturer
Department of Nursing&Midwifery
De Montfort University
Charles Frears Campus
266,London Road
Leicester
LE1 2RQ.

Named Clinician:Mr WM Harper,
Consultant Orthopaedic Surgeon.
Department of Orthopaedics
LRI NHS Trust.
Level 5
Balmoral Building
LRI.

4. Purpose of Research

4.1 Please state the objectives of the research and **briefly and simply** describe the scientific background.

Background.

Nursing assessment is a domain of clinical practice that is undergoing considerable change and development.Some measuring instruments such as Pain scales, Pressure sore risk assessment calculators, Glasgow coma scale and the Barthel Index,to name a few, enable nurses to carry out and objective,systematic and comprehensive nursing assessment.

Nurses who are in the forefront of care delivery have a clear role in the primary prevention and management of Deep Vein Thrombosis(DVT).A blanket nursing assessment of "Potential problem of DVT" provides a very crude index of measurement. The Autar DVT Scale (1994) was developed to:

- enable a systematic and objective risk assessment of clients at risk of DVT.
- identify patients at risk of DVT.
- calculate DVT risk in an individual in the context of multifactorial aetiology.
- provide quantifiable data for evaluation of practice.
- facilitate understanding of preventative and therapeutic practices of DVT.

Following formal approval by the Leicestershire Ethic Committee

(1994) a small study was undertaken to evaluate the DVT scale. It yielded some interesting and promising results in terms of the consistency and sensitivity of the instrument. However,the small scale study limits the generalisation of the results.

Objectives of proposed plan of Study

- to evaluate the consistency,sensitivity,specificity and predictive validity of the DVT scale in a large clinical population,to enable generalisation of findings.
- to evaluate the practical application of the DVT Scale.
- to explore current preventative practices in relation to the management of DVT.

5. Design of Study

5.1 Please describe what will be done, what results you expect and how you will analyse the results. Remember to attach a full copy of the protocol. Please identify potential dangers, discomfort or inconvenience to subjects of any of the techniques involved.

The DVT risk assessment scale is designed to enable its practical application in diverse clinical areas,where DVT is a problem.For this particular reason,this proposed research project is planned in the following clinical areas:

2 Orthopaedic Wards

1 General Surgical Ward

2 Integrated Medical Wards.

It is anticipated that a minimum of 150 patients will be recruited to the study from the above diverse clinical areas.Identification of those who are at risk as well as those who are not are equally important for targeting limited resources.Therefore, in the selected specialities, all patients carry some varying degree of risk and for this reason,sample randomisation is unnesssary and any patient admitted will be a prospective subject.

This will be essentially a data generating and quantitative study to test the consistency, sensitivity, specificity and practicality of the Autar DVT scale.Patients admitted to the negotiated diverse clinical areas will be risk assessed for DVT within 24 hours of admission, using the DVT scale.In order to ascertain that patients confidentiality is

maintained in its totality,only the ward nurses working in the negotiated areas,will be approached to access data on their patients. Data generated by staff mix of nurses will be analysed to determine the consistency of the DVT scale.As in the previous study (1994) nurses will be recruited to the study.The nursing record of each of the patients admitted to the study will be used to record the items in the DVT scale.Two registered nurses will independently complete a record on each patient,in order to provide comparative data,which will then be used to determine the consistency of this nursing assessment tool. For the consistency study, total percentage agreement (T%) and Kappa statistic will be applied.

Available data will also be translated into sensitivity and specificity of the instrument. Patients'scores will be analysed in terms of the optimal cutoff score for predictive accuracy.

Cross referencing the analysed data in relation to the 3 risk categories(low,moderate and high) with the patient's prescription chart,will provide an overview of the current therapeutic and prophylactic management strategy of DVT.

To evaluate the practicality of the DVT scale,a questionnaire will be applied to the DVT scale users.

As this is essentially a data generating study,it will not in anyway interfere with or alter the management of patients .The study will be undertaken after access has been duly negotiated and authorisation of the responsible nurse managers and consultants obtained. Colleagues (registered nurses) will be recruited on a voluntary basis and a programme of induction given to familiarise them with the DVT risk assessment scale.

6. Specific Details on Purpose and Design of Study

6.1 Has this work been carried out before? Yes
If yes, why is there a need to repeat?
The very small study limits the application and generalisation of findings.

6.2 Is this a multi-centre study? Yes

6.3 What type of a study is this (tick **one** as appropriate):

Pilot	
Definitive	
Follow on of a previous study	YES
Modification of a previous study	

If a follow on or modification of a previous study, please give reference number of previous application and approval date:

Previous study reference number : AEC\MS
Approval date:6th June 1994.

N.B. For the next two sections circle one as appropriate.

6.4 Is this research of direct benefit to the subject: Yes

6.5 Is this research related to: **D**iagnosis. No
If yes to therapy then what type (tick **any** as appropriate):

Drugs	
Surgery	
Other	

7. New Chemical Entity/Therapeutic Agent or Established Agent

NOT APPLICABLE

7.1 Is this an investigation of an **established** agent? No
If yes, is the established agent being used for a new/unlicensed indication?
Yes/No

7.2 Is this a study of a **new** chemical entity/therapeutic agent? No
If yes, What stage is this in its evaluation? (tick **one** as appropriate)

Phase II	
Phase III	
Phase IV	

7.3 If this drug is being supplied by a pharmaceutical company as part of sponsored research, has a clinical trial certificate/exemption certificate been provided?
Yes/No

8. Substances to be Administered

NOT APPLICABLE

	Route	Amount/ Frequency	Risks	Precautions
Drugs*				
Isotopes				
Fluids & Diets				
Others				

* On completion of a clinical trial the investigator should notify the pharmacist who will destroy any chemical trial material still being held.

9. Financial Arrangements

NOT APPLICABLE

9.1 Will you be receiving any financial contributions towards your research?
No
If yes, from whom and how much?
N.B. Tick main source only. NOT APPLICABLE

Whom:
- Pharmaceutical Company ☐
- Research Grant ☐
- (MRC, BHF, RHA e.t.c)
- Other ☐

How much? (Please state approx total amount): **£**

9.2 How will these funds be spent? (tick **any** as appropriate)
- Staff costs ☐
- Running Costs ☐
- Other ☐

If 'other' briefly describe:

10. Recruitment of Subjects

Please say how you will recruit subjects, with rules of inclusion and exclusion and any proposals to deny and delay treatment and any other relevant details e.g. age, sex, type of patient.
N.B. Investigators are reminded of the need to notify General Practitioners when patients under their immediate care are to be included in a study. Investigators should ensure that any patients or healthy volunteers involved in a particular project are not included in another study which also involves drugs or isotopes.

10.1 Data access will be from a convenient sample of 150 patients across the diverse clinical specialities.

10.2 Controls? Not applicable.

10.3 Number of subjects to be recruited: 150 subjects across the specialities for data collection by at least two registered nurses(volunteers) from each of the negotiated wards.

10.4 Type of subjects to be recruited? (tick **any** as appropriate)

Patients	/
Volunteers	/

If volunteers, then please tick **any** of the options from the list below to specify the type:

Staff	/
Student	
Other	

10.5 Any financial inducements offered to subjects or relatives?No
If yes, what?

10.6 **Informed Consent**
N.B. Written informed consent is preferred, ***a copy of the consent form and information leaflet to be used must be supplied.***
Please note that special consideration must be given to children, mentally ill and handicapped.

Will informed consent be obtained? Yes
If yes, then tick **any** of the options from the list below to specify the method used:

Written:	
Oral	/

10.7 **Compensation NOT APPLICABLE**

What kind of arrangements for compensation/indemnity for subjects are in place for the study, please tick **any** of the options from the list below:

ABPI Guidelines on compensation for medicine induced injury	
Trust Indemnity	
Other	

If yes to 'Other', please give details:

10.8 **Investigations of Subjects/Controls**: NOT APPLICABLE

Venous samples	Yes/No
If yes, then	Where From:
	Frequency:
	Amount:

Arterial samples	Yes/No
If yes, then	Where From:
	Frequency:
	Amount:

N.B. If yes to any of the following questions, please give details below.

X-rays	Yes/No
Radiation	Yes/No
Ultrasonics	Yes/No

Biopsies	Yes/No
If yes, then	Site:
	Method:
	Size:
	Number:

Anaesthesia	Yes/No
If yes, then	Local or General

Other invasions Cannulae/ Probes/ Catheters/ Endoscopes/ Lumbar Punctures

Any non-invasive tests? EEG/EMG/ECG

Psychological tests? Yes/No

Questionnaires? Yes/No
If yes, ***please include copy.***

Other Activities Yes/No

Additional Details:

11. Likely Benefits Of Study

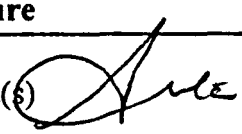
The DVT scale could provide consistency in the assessment of patients. It has the potential for development into a predictive index for DVT risk. Formalising the assessment of DVT could improve uptake of accurate venous thromboembolism prophylaxis(Byrne et al 1996).

12. Documents Enclosed

Please remember to enclose the following documents where appropriate:

- Detailed Protocol (DVT Scale) Yes
- Questionnaire Yes
- Proposed Consent Form No
- Patient Information Leaflet No
- CTX No
- Confirmation of compensation arrangements No

13. Applicant(s) Signature

Signature(s) of Applicant(s)  Date: 26/2/97

14. Countersignature of Consultant

Countersignature of Consultant (in the case of junior medical and dental staff), Head of Department, Nurse Tutor, Director of Nursing Services etc.

I have discussed the research proposal with the investigator, who is in my department, and I support his/her application to the Ethical Committee.

Signature(s) ..... Date: 26/2/97

Please print name: Dr Stuart Brand .

.....

R&D Department Stamp

Appendix 3

Melanie Sursham
Direct Dial 0116 258 8610

Health for Leicestershire
LEICESTERSHIRE HEALTH
Gwendolen Road, Leicester LE5 4QF
Tel: (0116) 273 1173 Fax: (0116) 258 8577
DX 709470 Leicester 12

27 May 1997

Mr R Autar
Senior Lecturer in Biological Sciences & Nursing
De Montfort University
Charles Frears Campus
266 London Road
Leicester LE2 1RQ

Dear Mr Autar

Advancing Nursing Practice in the management of Deep Vein Thrombosis- Development - Application and Evaluation of the Autar DVT Scale - our ref. no. 4593

Further to your letters dated 29 April and 12 May, you will be pleased to know that the Leicestershire Ethics Committee at its meeting held on the 2 May, 1997 approved your request to undertake the above-mentioned research.

Your attention is drawn to the attached paper which reminds the researcher of information that needs to observed when ethics committee approval is given.

Yours sincerely

M. Sursham

R F Bing pp
Chairman
Leicestershire Ethics Committee

(NB All communications relating to Leicestershire Ethics Committee must be sent to the Committee Secretariat at Leicestershire Health)

Appendix 4

THE
LEICESTER ROYAL INFIRMARY
NHS TRUST
June 10, 1997

ARCH AND
LOPMENT

ARCH OFFICE

Windows
ch and
opment Support
er

16 258 6318
16 258 7558
@lri2.demon.co.uk

Kelley PhD
Allen

16 258 6642
16 258 6448
@lri2.demon.co.uk

CTOR OF
ARCH AND
LOPMENT

vor A. Howlett
RCP
1332@compuserve.com

Project Ref: RFD 863
Please use this reference in all correspondence with the research office, regarding this project.

Department of Nursing & Midwifery,
De Montfort University
Charles Frears Campus
266 London Road

Dear Autar,

Re: Advancing Nursing Practice in the Management of Deep Vein Thrombosis (DVT). Development, application and evaluation of the Autar DVT risk assessment scale

Your research notification form has been approved and your project is indemnified by the Trust.

Could you please notify this department if there are any changes to the commencement or end dates for the project.

Yours sincerely,

Dr Trevor A. Howlett, MD, FRCP.
Director of Research & Development

ADDRESS

Leicester
LE1 5WW

Telephone
16 254 1414

Facsimile
16 258 5631

Appendix 5.1

UNIVERSITY OF LEICESTER

SCHOOL OF MEDICINE · THE GLENFIELD HOSPITAL NHS TRUST
GROBY ROAD · LEICESTER LE3 9QP · ENGLAND

DEPARTMENT OF ORTHOPAEDIC SURGERY

HEAD OF DEPARTMENT
Professor P J GREGG
TELEPHONE
0116 256 3012

SENIOR LECTURERS
MR WM HARPER
MR P D TRIFFITT
TELEPHONE
0116 256 3050

MR H THOMAS (Hon)

NON-CLINICAL LECTURER
DR P ROONEY
TELEPHONE
0116 256 3049

RESEARCH OFFICE
TELEPHONE
0116 256 3019

FACSIMILE
0116 232 1702

WMH/jas/AUTAR

28 January 1997

Ricky Autar
Senior Lecturer in Nursing
School of Health and Community Studies
Department of Nursing and Midwifery
De Montfort University
Charles Frear Campus
266 London Road
LEICESTER LE2 1RQ

Dear Ricky

Thanks very much for your note and paper which I will read in due course. I am more than happy for you to have access to my patients on the orthopaedic level at the Leicester Royal Infirmary, and also my elective patients at the Glenfield Hospital. It may interest you to note that all my elective joint replacements have a venogram performed postoperatively.

May I wish you every success with your work.

Yours sincerely

W M Harper
Senior Lecturer/Hon. Consultant Orthopaedic Surgeon

THE QUEEN'S ANNIVERSARY PRIZES
1994

UNIVERSITY OF LEICESTER

FACULTY OF MEDICINE AND BIOLOGICAL SCIENCES
ROBERT KILPATRICK CLINICAL SCIENCES BUILDING · LEICESTER ROYAL INFIRMARY
P O BOX 65 · LEICESTER LE2 7LX · ENGLAND

DIVISION OF MEDICINE & THERAPEUTICS
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HT/SW

5th October 1999

Ricky Autar
Senior Lecturer in Biological Sciences & Nursing
De Montfort University
Charles Frears Campus
266 London Road
Leicester LE2 1RQ

Dear Ricky

Re: DVT Risk Assessment Study

Thank you for your letter defining a few details of your study, and sending me a copy of your paper on the Autar DVT scales.

I am quite happy for any of my patients to take part in the study and I await the results with interest.

Yours sincerely

Bert Thurston

H Thurston
Professor of Medicine

Appendix 5.3

ADNS/CJ

THE GLENFIELD HOSPITAL NHS TRUST
Groby Road
Leicester LE3 9QP
Tel (0116) 287 1471
Fax (0116) 258 3950

DEPARTMENT OF GENERAL SURGERY

18th March 1999

Mr R Autar,
Senior Lecturer in Biological Science & Nursing,
De Montfort University,
Charles Frears Campus,
266 London road,
Leicester,
LE2 1RQ.

Dear Mr Autar,

Many thanks for your letter of March 12th concerning the proposed study on DVT risk assessment. I remain happy for data from the Surgical Unit to be used for this study.

However, I remain uneasy as to the amount of input required from nursing staff on the ward. This seems to involve not only completing the risk assessment itself but also maintaining a coded register of patients and, most worryingly of all, obtaining the consent of patients to be included in the study. Conventionally it is the researcher who takes responsibility for obtaining consent and carrying out the data collection and I do not have the authority to sanction the use of ward staff in this way. I have asked Ruth Chapman, the Business Manager in Surgery, to discuss this with the Department of Nursing and the ward nurses in order to ascertain the feasibility and practicality of this study.

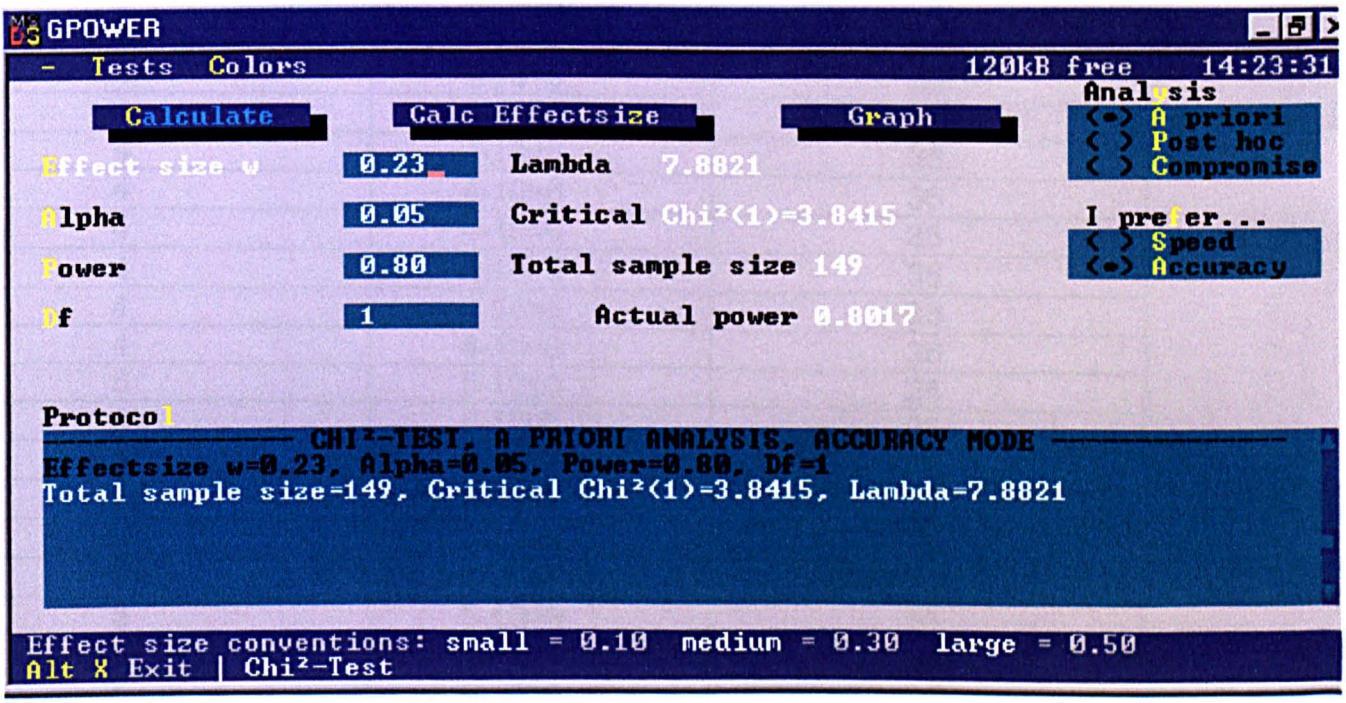
With best wishes,

Yours sincerely,

A D N SCOTT BSc MS FRCS
Clinical Chairman

c.c. Mrs R Chapman,
Assistant Specialty Manager - Surgical Services,
Glenfield Hospital.

Appendix 6
Sample size : apriori calculation



Appendix 7

Standard Deviation of the risk assessment score & Standard Error of Mean

Mean= 10

N= 141

Df= 1

Observations (x)	Deviations ($x-\bar{x}$)	Squared deviations $(x-\bar{x})^2$
1	1-10=9	81
1	1-10=9	81
2	2-10=8	64
2	2-10=8	64
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
4	4-10=6	36
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
5	5-10=5	25
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16
6	6-10=4	16

7	7-10=3	9
7	7-10=3	9
7	7-10=3	9
7	7-10=3	9
7	7-10=3	9
7	7-10=3	9
7	7-10=3	9
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
8	8-10=2	4
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9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
9	9-10=1	1
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
10	10-10=0	0
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1

11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
11	11-10=1	1
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
12	12-10=2	4
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
13	13-10=3	9
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14	14-10=4	16
14	14-10=4	16
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15	15-10=5	25
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16	16-10=6	36
16	16-10=6	36
16	16-10=6	36
16	16-10=6	36
16	16-10=6	36
17	17-10=7	49
17	17-10=7	49
17	17-10=7	49
19	19-10=9	81

20	20-10=10	100
20	20-10=10	100
20	20-10=10	100
26	26-10=16	256
27	27-10=17	289
Total= 141		$\sum$= 2865

$$S^2 = \frac{2865}{141-1} = 20.4642$$

$$SD = \sqrt{20.4642} = 4.5237 = \doteq 5$$

$$SEM = \frac{SD}{\sqrt{N}} \frac{5}{141} = \doteq 0.04$$

The SD of 5 measured the variability among the individual risk assessment scores, SEM calculated the variability among the samples.

ogistic Regression(orthopaedics) appendix 8

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	50	100.0
	Missing Cases	0	.0
	Total	50	100.0
Unselected Cases		0	.0
Total		50	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
0	0
1	1

Block 0: Beginning Block

Classification Table[a,b]

			Predicted		
			DVT		Percentage Correct
	Observed		0	1	
Step 0	DVT	0	42	0	100.0
		1	8	0	.0
	Overall Percentage				84.0

a. Constant is included in the model.

b. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 0	Constant	-1.658	.386	18.478	1	.000	.190

Variables not in the Equation

			Score	df	Sig.
Step 0	Variables	AGE	4.801	1	.028
		MOBILITY	3.930	1	.047
		BUILD	6.042	1	.014
		TRAUMA	8.470	1	.004
		SURGERY	3.554	1	.059
		DISEASES	.303	1	.582
		TOTAL	6.275	1	.012
	Overall Statistics		14.907	7	.037

Block 1: Method = Forward Stepwise (Wald)

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	11.369	1	.001
	Block	11.369	1	.001
	Model	11.369	1	.001

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	32.598	.203	.348

Hosmer and Lemeshow Test

Step	Chi-square	df	Sig.
1	.001	1	.977

Contingency Table for Hosmer and Lemeshow Test

		DVT = 0		DVT = 1		
		Observed	Expected	Observed	Expected	Total
Step 1	1	18	18.000	0	.000	18
	2	1	.999	0	.001	1
	3	23	23.000	8	8.000	31

Classification Table[a]

			Predicted		
			DVT		Percentage Correct
	Observed		0	1	
Step 1	DVT	0	42	0	100.0
		1	7	1	12.5
	Overall Percentage				86.0

a. The cut value is .500

Variables in the Equation[b]

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	TRAUMA	5.942	35.392	.028	1	.867	380.553
	Constant	-24.956	141.562	.031	1	.860	.000

Variables in the Equation[b]

		95.0% C.I.for EXP(B)	
		Lower	Upper
Step 1[a]	TRAUMA	.000	5.08E+32
	Constant		

a. Variable(s) entered on step 1: TRAUMA.

b. Stepwise procedure stopped because removing the least significant variable result in a previously fitted model.

Correlation Matrix

		Constant	TRAUMA
Step 1	Constant	1.000	-1.000
	TRAUMA	-1.000	1.000

Variables not in the Equation[a]

			Score	df	Sig.
Step 1	Variables	AGE	1.936	1	.164
		MOBILITY	.528	1	.467
		BUILD	4.635	1	.031
		SURGERY	.000	1	1.000
		DISEASES	.137	1	.711
		TOTAL	.559	1	.455

a. Residual Chi-Squares are not computed because of redundancies.

```
Step number: 1

Observed Groups and Predicted Probabilities

                32 +                                                  +
                   |                    1                           |
                   |                    1                           |
                   |                    1                           |
F               24 +                    0                           +
R                  |                    0                           |
E                  |0                   0                           |
Q                  |0                   0                           |
U               16 +0                   0                           +
E                  |0                   0                           |
N                  |0                   0                           |
C                  |0                   0                           |
Y                8 +0                   0                           +
                   |0                   0                           |
                   |0                   0                           |
                   |0                   0                           |
                   |0                   0                           1
Predicted          +---------+----------+----------+----------+-------+
  Prob:            0        .25        .5        .75        1
  Group:           00000000000000000000000000000011111111111111111111111111111

         Predicted Probability is of Membership for 1
         The Cut Value is .50
         Symbols: 0 - 0
                  1 - 1
```

Each Symbol Represents 2 Cases.

Casewise List[b]

Case	Selected Status[a]	Observed DVT	Predicted	Predicted Group	Temporary Variable Resid	Temporary Variable ZResid
4	S	0	.001	0	-.001	-.028

a. S = Selected, U = Unselected cases, and ** = Misclassified cases.

b. Cases with studentized residuals greater than 2.000 are listed.

Logistic Regression (Medical) Appendix 9

Case Processing Summary

Unweighted Cases[b]		N	Percent
Selected Cases[a]	Included in Analysis	50	100.0
	Missing Cases	0	.0
	Total	50	100.0
Unselected Cases		0	.0
Total		50	100.0

a. The variable TRAUMA is constant for all selected cases. Since a constant was requested in the model, it will be removed from the analysis.

b. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
0	0
1	1

Block 0: Beginning Block

Classification Table[a,b]

Observed			Predicted		
			DVT		Percentage Correct
			0	1	
Step 0	DVT	0	38	0	100.0
		1	12	0	.0
	Overall Percentage				76.0

a. Constant is included in the model.

b. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 0	Constant	-1.153	.331	12.117	1	.000	.316

Variables not in the Equation

			Score	df	Sig.
Step 0	Variables	AGE	.048	1	.827
		BUILD	8.416	1	.004
		MOBILITY	.163	1	.687
		SPECIAL	.322	1	.570
		TOTAL	1.176	1	.278
	Overall Statistics		9.145	5	.103

Block 1: Method = Enter

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	9.492	5	.091
	Block	9.492	5	.091
	Model	9.492	5	.091

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	45.616	.173	.259

Hosmer and Lemeshow Test

Step	Chi-square	df	Sig.
1	3.698	7	.814

Contingency Table for Hosmer and Lemeshow Test

		DVT = 0		DVT = 1		
		Observed	Expected	Observed	Expected	Total
Step 1	1	6	5.814	0	.186	6
	2	4	4.545	1	.455	5
	3	5	4.316	0	.684	5
	4	6	4.968	0	1.032	6
	5	6	6.470	2	1.530	8
	6	3	3.142	1	.858	4
	7	3	3.719	2	1.281	5
	8	3	3.000	2	2.000	5
	9	2	2.025	4	3.975	6

Classification Table[a]

			Predicted		
			DVT		Percentage Correct
	Observed		0	1	
Step 1	DVT	0	36	2	94.7
		1	8	4	33.3
	Overall Percentage				80.0

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	AGE	.069	.599	.013	1	.908	1.072
	BUILD	1.479	.618	5.721	1	.017	4.388
	MOBILITY	-.369	.450	.670	1	.413	.692
	SPECIAL	-1.851	12.231	.023	1	.880	.157
	TOTAL	.083	.140	.350	1	.554	1.086
	Constant	-3.611	2.340	2.380	1	.123	.027

Variables in the Equation

		95.0% C.I.for EXP(B)	
		Lower	Upper
Step 1[a]	AGE	.331	3.466
	BUILD	1.306	14.741
	MOBILITY	.286	1.672
	SPECIAL	.000	4.05E+09
	TOTAL	.826	1.428
	Constant		

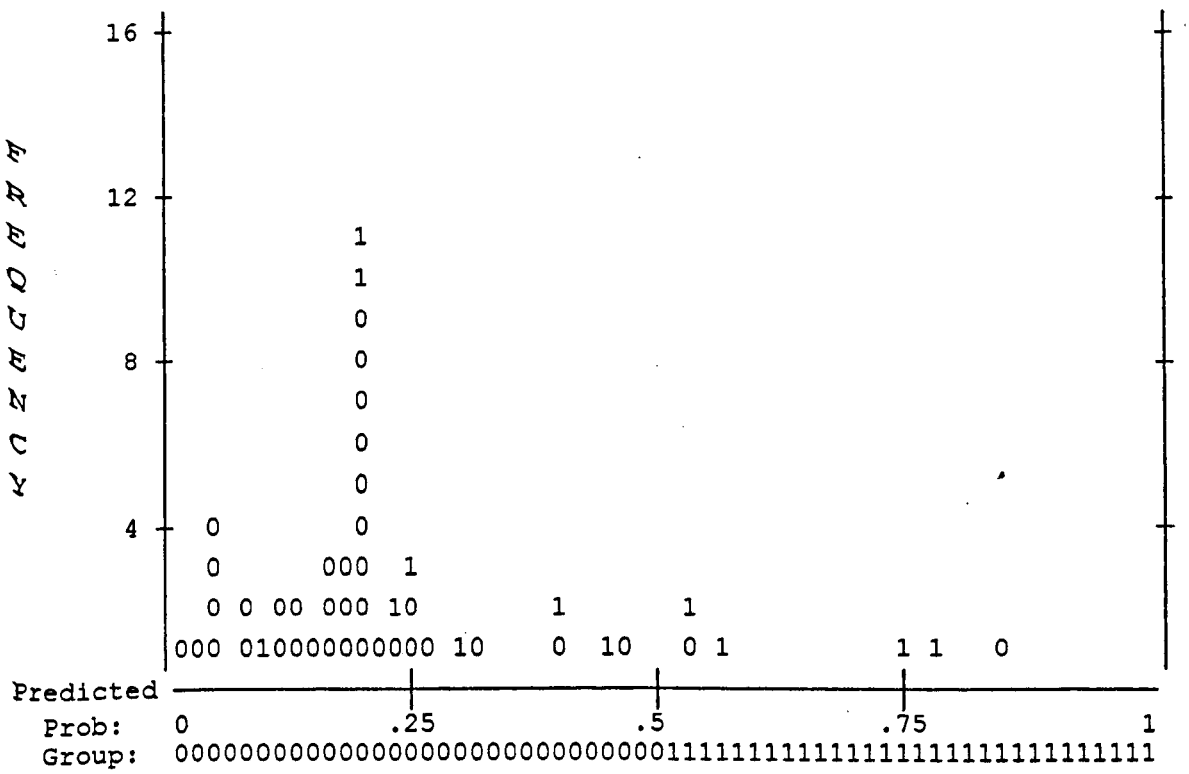
a. Variable(s) entered on step 1: AGE, BUILD, MOBILITY, SPECIAL, TOTAL.

Correlation Matrix

		Constant	AGE	BUILD	MOBILITY	SPECIAL	TOTAL
Step 1	Constant	1.000	-.861	-.454	.200	-.041	-.058
	AGE	-.861	1.000	.215	.016	.041	-.330
	BUILD	-.454	.215	1.000	-.299	.010	-.096
	MOBILITY	.200	.016	-.299	1.000	.003	-.584
	SPECIAL	-.041	.041	.010	.003	1.000	-.008
	TOTAL	-.058	-.330	-.096	-.584	-.008	1.000

Step number: 1

Observed Groups and Predicted Probabilities



Predicted Probability is of Membership for 1
The Cut Value is .50
Symbols: 0 - 0
1 - 1
Each Symbol Represents 1 Case.

Casewise List[b]

Case	Selected Status[a]	Observed DVT	Predicted	Predicted Group	Temporary Variable Resid	Temporary Variable ZResid
25	S	1**	.088	0	.912	3.220
44	S	0**	.844	1	-.844	-2.324

a. S = Selected, U = Unselected cases, and ** = Misclassified cases.

b. Cases with studentized residuals greater than 2.000 are listed.

Logistic Regression (surgical directorate) appendix 10.

Case Processing Summary

Unweighted Cases[c]		N	Percent
Selected Cases[a,b]	Included in Analysis	50	100.0
	Missing Cases	0	.0
	Total	50	100.0
Unselected Cases		0	.0
Total		50	100.0

a. The variable Special risk is constant for all selected cases. Since a constant was requested in the model, it will be removed from the analysis.

b. The variable TRAUMA is constant for all selected cases. Since a constant was requested in the model, it will be removed from the analysis.

c. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
No DVT	0
DVT	1

Block 0: Beginning Block

Classification Table[a,b]

			Predicted		
			DVT		Percentage Correct
	Observed		No DVT	DVT	
Step 0	DVT	No DVT	42	0	100.0
		DVT	8	0	.0
	Overall Percentage				84.0

a. Constant is included in the model.

b. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 0	Constant	-1.658	.386	18.478	1	.000	.190

Variables not in the Equation

			Score	df	Sig.
Step 0	Variables	AGE	1.553	1	.213
		BUILD	.846	1	.358
		MOBILITY	.131	1	.717
		SURGERY	.027	1	.868
		HIGH_RIS	9.433	1	.002
	Overall Statistics		15.236	5	.009

Block 1: Method = Forward Stepwise (Wald)

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	8.184	1	.004
	Block	8.184	1	.004
	Model	8.184	1	.004
Step 2	Step	5.695	1	.017
	Block	13.879	2	.001
	Model	13.879	2	.001

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	35.783	.151	.258
2	30.088	.242	.414

Hosmer and Lemeshow Test

Step	Chi-square	df	Sig.
1	.411	2	.814
2	4.328	6	.632

Contingency Table for Hosmer and Lemeshow Test

		DVT = No DVT		DVT = DVT		
		Observed	Expected	Observed	Expected	Total
Step 1	1	30	29.969	2	2.031	32
	2	5	5.035	1	.965	6
	3	3	3.515	2	1.485	5
	4	4	3.480	3	3.520	7
Step 2	1	5	4.983	0	.017	5
	2	5	4.946	0	.054	5
	3	5	5.811	1	.189	6
	4	2	1.829	0	.171	2
	5	17	16.283	1	1.717	18
	6	4	4.279	1	.721	5
	7	3	2.854	2	2.146	5
	8	1	1.015	3	2.985	4

Classification Table[a]

			Predicted		
			DVT		Percentage Correct
	Observed		No DVT	DVT	
Step 1	DVT	No DVT	42	0	100.0
		DVT	7	1	12.5
	Overall Percentage				86.0
Step 2	DVT	No DVT	41	1	97.6
		DVT	4	4	50.0
	Overall Percentage				90.0

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1[a]	HIGH_RIS	.380	.143	7.097	1	.008	1.463
	Constant	-2.692	.660	16.614	1	.000	.068
Step 2[b]	SURGERY	1.139	.558	4.164	1	.041	3.122
	HIGH_RIS	.720	.242	8.865	1	.003	2.053
	Constant	-5.665	1.774	10.201	1	.001	.003

Variables in the Equation

		95.0% C.I.for EXP(B)	
		Lower	Upper
Step 1[a]	HIGH_RIS	1.106	1.935
	Constant		
Step 2[b]	SURGERY	1.046	9.319
	HIGH_RIS	1.279	3.298
	Constant		

a. Variable(s) entered on step 1: HIGH_RIS.

b. Variable(s) entered on step 2: SURGERY.

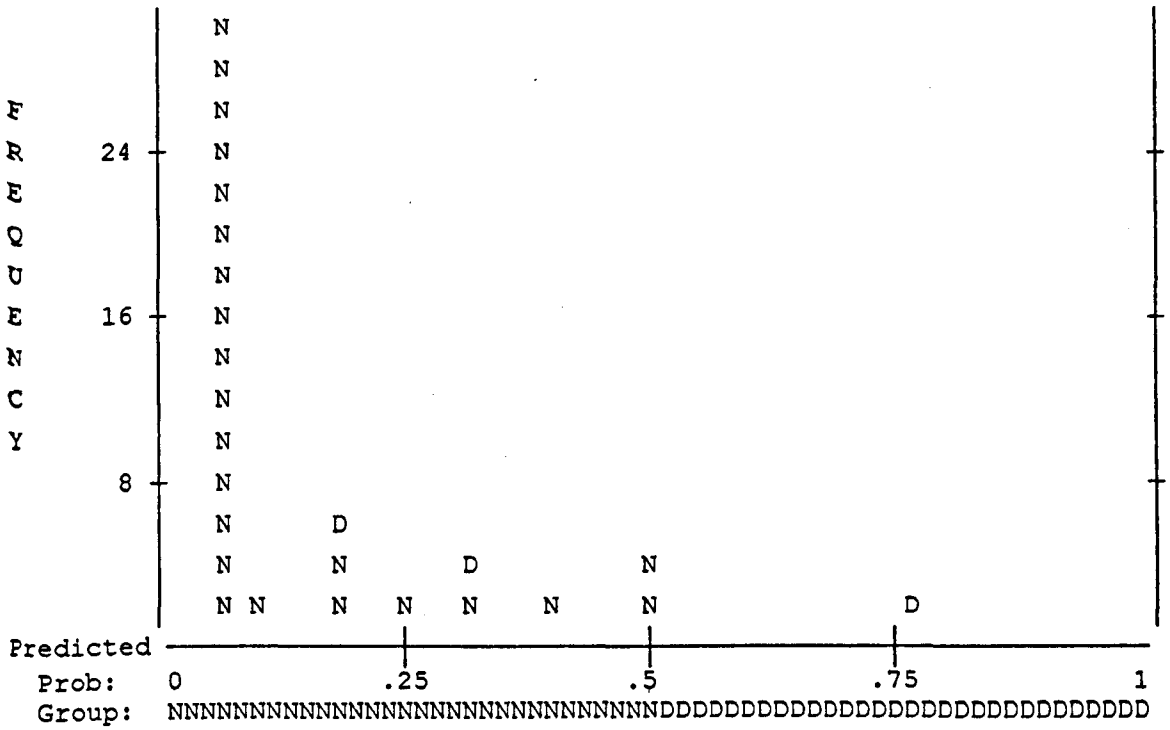
Correlation Matrix

		Constant	HIGH_RIS	SURGERY
Step 1	Constant	1.000	-.759	
	HIGH_RIS	-.759	1.000	
Step 2	Constant	1.000	-.894	-.921
	SURGERY	-.921	.776	1.000
	HIGH_RIS	-.894	1.000	.776

Variables not in the Equation

			Score	df	Sig.
Step 1	Variables	AGE	.341	1	.559
		BUILD	2.257	1	.133
		MOBILITY	.113	1	.736
		SURGERY	4.958	1	.026
	Overall Statistics		7.453	4	.114
Step 2	Variables	AGE	.047	1	.828
		BUILD	3.794	1	.051
		MOBILITY	.246	1	.620
	Overall Statistics		4.554	3	.208

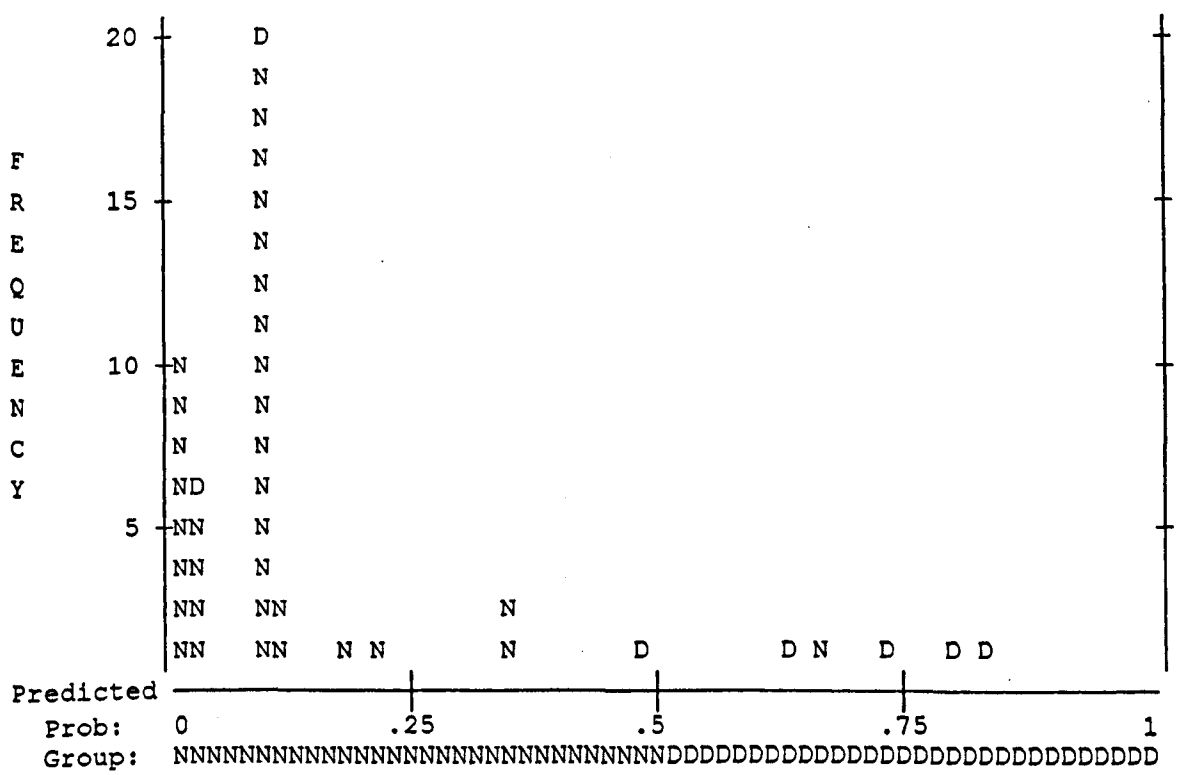
```
Step number: 1

Observed Groups and Predicted Probabilities

32 +    D                                                              +
```



Predicted Probability is of Membership for DVT
The Cut Value is .50
Symbols: N - No DVT
D - DVT
Each Symbol Represents 2 Cases.

Step number: 2

Observed Groups and Predicted Probabilities



Predicted Probability is of Membership for DVT
The Cut Value is .50

Symbols: N - No DVT
D - DVT
Each Symbol Represents 1.25 Cases.

Casewise List[b]

Case	Selected Status[a]	Observed DVT	Predicted	Predicted Group	Temporary Variable Resid	Temporary Variable ZResid
2	S	D**	.033	N	.967	5.441
5	S	D**	.095	N	.905	3.079
39	S	D**	.112	N	.888	2.811

a. S = Selected, U = Unselected cases, and ** = Misclassified cases.

b. Cases with studentized residuals greater than 2.000 are listed.

ROC Curve: Orthopaedics: Appendix 11

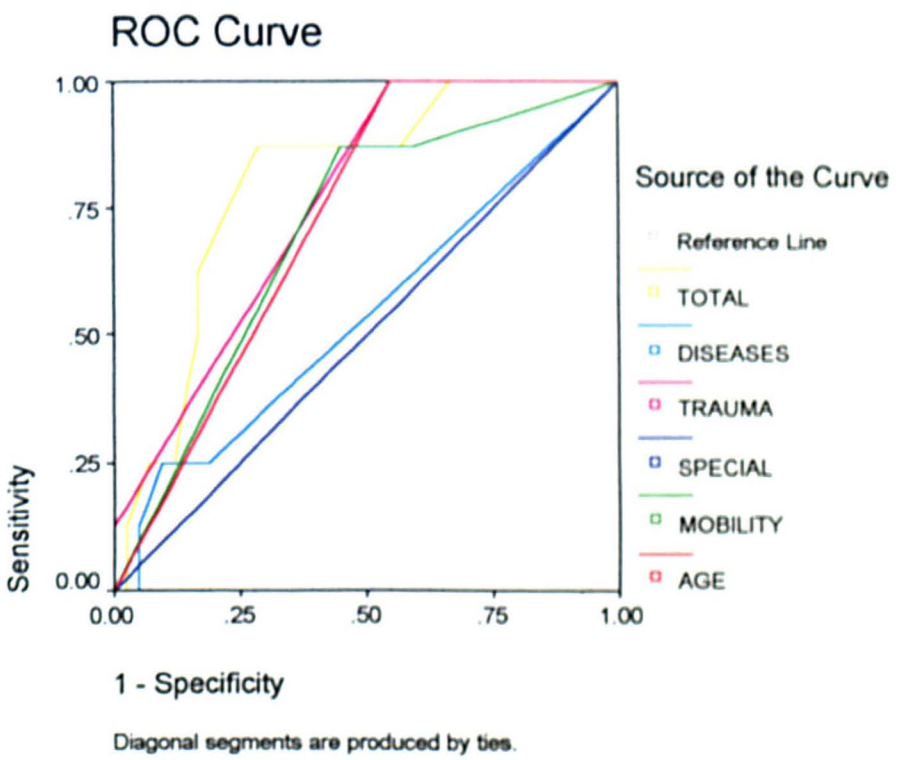
Case Processing Summary

DVT[b]	Valid N (listwise)
Positive[a]	8
Negative	42

Larger values of the test result variable(s) indicate stronger evidence for a positive actual state.

a. The positive actual state is 1.

b. The test result variable(s): TRAUMA has at least one tie between the positive actual state group and the negative actual state group.



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
AGE	.726	.077	.044	.576	.877
MOBILITY	.702	.094	.072	.518	.887
SPECIAL	.500	.112	1.000	.280	.720
TRAUMA	.760	.077	.021	.610	.911
DISEASES	.539	.117	.731	.308	.769
TOTAL	.801	.076	.008	.652	.949

The test result variable(s): AGE, MOBILITY, SPECIAL, TRAUMA, DISEASES, TOTAL has at least c tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Coordinates of the Curve

Test Result Variable(s)	Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity
AGE	-1.00	1.000	1.000
	.50	1.000	.857
	1.50	1.000	.690
	2.50	1.000	.619
	3.50	1.000	.548
	5.00	.000	.000
MOBILITY	-1.00	1.000	1.000
	.50	.875	.595
	1.50	.875	.548
	2.50	.875	.476
	3.50	.875	.452
	5.00	.000	.000
SPECIAL	-1.00	1.000	1.000
	1.00	.000	.000
TRAUMA	-1.00	1.000	1.000
	1.50	1.000	.571
	3.50	1.000	.548
	6.50	.125	.000
	10.00	.000	.000
DISEASES	-1.00	1.000	1.000
	1.00	.250	.190
	2.50	.250	.167
	4.00	.250	.143
	6.00	.250	.095
	7.50	.125	.048
	9.50	.000	.048
	11.50	.000	.024
	13.00	.000	.000
TOTAL	1.00	1.000	1.000
	3.00	1.000	.952
	4.50	1.000	.929
	5.50	1.000	.881
	6.50	1.000	.738
	7.50	1.000	.690
	8.50	1.000	.667
	9.50	.875	.571
	10.50	.875	.500
	11.50	.875	.381

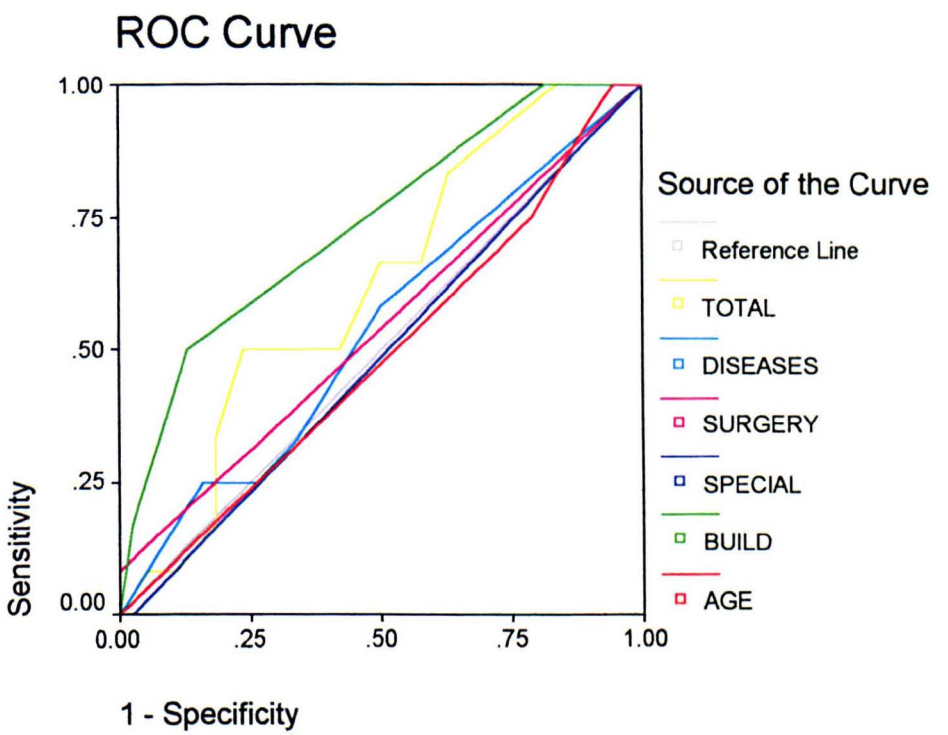
ROC Curve (medical directorate) Appendix 12.

Case Processing Summary

DVT	Valid N (listwise)
Positive [a]	12
Negative	38

Larger values of the test result variable(s) indicate stronger evidence for a positive actual state.

a. The positive actual state is 1.



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
AGE	.487	.096	.892	.299	.675
BUILD	.735	.083	.015	.571	.898
SPECIAL	.487	.095	.892	.300	.674
SURGERY	.542	.100	.666	.346	.737
DISEASES	.537	.097	.699	.348	.726
TOTAL	.620	.087	.216	.448	.791

The test result variable(s): AGE, BUILD, SPECIAL, SURGERY, DISEASES, TOTAL has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Coordinates of the Curve

Test Result Variable(s)	Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity
AGE	-1.00	1.000	1.000
	.50	1.000	.974
	1.50	1.000	.947
	2.50	.917	.895
	3.50	.750	.789
	5.00	.000	.000
BUILD	-1.00	1.000	1.000
	.50	1.000	.816
	1.50	.500	.132
	2.50	.167	.026
	4.00	.000	.000
SPECIAL	-1.00	1.000	1.000
	1.50	.000	.026
	4.00	.000	.000
SURGERY	-1.00	1.000	1.000
	.50	.083	.000
	2.00	.000	.000
DISEASES	-1.00	1.000	1.000
	1.50	.583	.500
	3.50	.333	.342
	4.50	.250	.263
	6.00	.250	.158
	8.00	.000	.000
TOTAL	.00	1.000	1.000
	2.50	1.000	.974
	4.50	1.000	.842
	5.50	.833	.632
	6.50	.667	.579
	7.50	.667	.500
	8.50	.500	.421
	9.50	.500	.342
	10.50	.500	.237
	11.50	.333	.184
	12.50	.167	.184
	13.50	.083	.105
	14.50	.083	.079
	15.50	.083	.053
	17.00	.000	.000

The test result variable(s): AGE, BUILD, SPECIAL, SURGERY, DISEASES, TOTAL has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

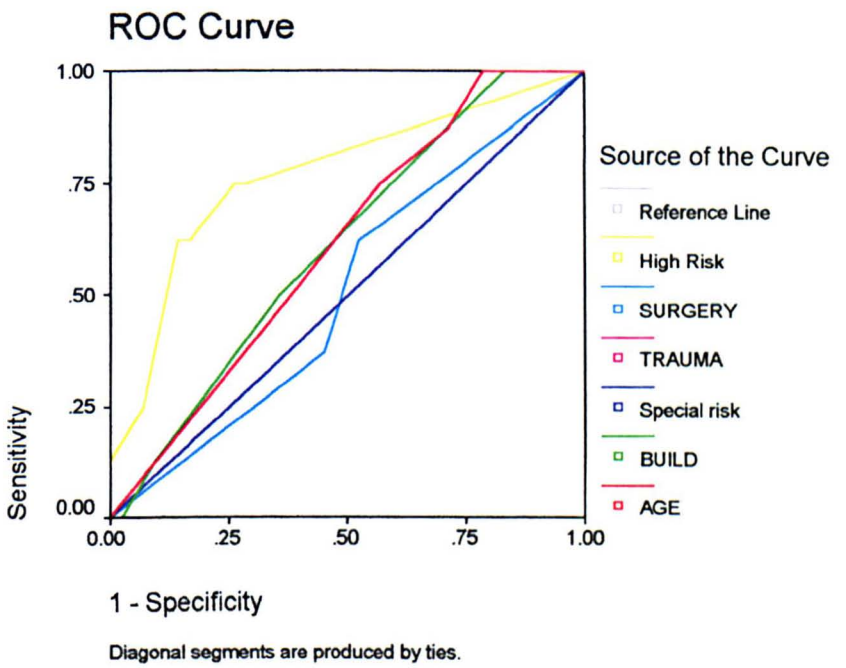
ROC Curve: (surgical directorate) Appendix 13

Case Processing Summary

DVT	Valid N (listwise)
Positive[a]	8
Negative	42

Larger values of the test result variable(s) indicate stronger evidence for a positive actual state.

a. The positive actual state is DVT.



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
AGE	.612	.097	.321	.422	.801
BUILD	.610	.098	.328	.417	.803
Special risk	.500	.112	1.000	.280	.720
TRAUMA	.500	.112	1.000	.280	.720
SURGERY	.506	.106	.958	.299	.713
High Risk	.768	.100	.017	.571	.965

The test result variable(s): AGE, BUILD, Special risk, TRAUMA, SURGERY, High Risk has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Coordinates of the Curve

Test Result Variable(s)	Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity
AGE	-1.00	1.000	1.000
	.50	1.000	.929
	1.50	1.000	.786
	2.50	.875	.714
	3.50	.750	.571
	5.00	.000	.000
BUILD	-1.00	1.000	1.000
	.50	1.000	.833
	1.50	.500	.357
	2.50	.125	.095
	3.50	.000	.024
	5.00	.000	.000
Special risk	-1.00	1.000	1.000
	1.00	.000	.000
TRAUMA	-1.00	1.000	1.000
	1.00	.000	.000
SURGERY	-1.00	1.000	1.000
	.50	.750	.690
	1.50	.625	.524
	2.50	.375	.452
	4.00	.000	.000
High Risk	-1.00	1.000	1.000
	.50	.750	.286
	2.00	.750	.262
	3.50	.625	.167
	4.50	.625	.143
	5.50	.375	.095
	6.50	.250	.071
	8.50	.125	.000
	11.00	.000	.000

The test result variable(s): AGE, BUILD, Special risk, TRAUMA, SURGERY, High Risk has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

Appendix 14

Data Analysis: DVT scale questionnaires (Trauma Directorate).

Total No of postal questionnaire:13 (100%). Total No of respondents:12 (92%).

Respondents

Item No	1	2	3	4	5	6	7	8	9	10	11	12
1	21yrs	< 6 mts	6..5 mts	14yrs	2yrs	30yrs	18yrs	33yrs	2.5yrs	6yrs	8yrs	11yrs
2	EN	RGN DIP	RGN ENB	RGN	RGN| DIP	RGN ONC	RGN ONC	RGN DIP	RGN DIP	RGN ENB	RGN ENB	RGN ENB
3	D	D	F	F	E	D	D	D	E	E	E	F
4	21yrs	< 6 mts	7yrs	14 mts	18mts	2yrs	18mts	<6mts	18mts	18mts	8yrs	11yrs.
5	No	No	No	No	No	No	No	No	No	No	No	No
6	<5	11-15	<5	<5	<5	5-10	<5	11-15	11-15	11-15	5-10	<5
7.1	2	5	4	4	4	4	5	3	4	4	4	4
7.2	4	5	4	4	4	4	4	3	4	4	4	4
7.3	4	4	4	4	4	4	5	4	4	5	4	4
7.4	4	4	4	4	4	4	3	4	3	4	3	4
8	4	5	4	4	4	4	4	4	5	4	4	4
9	5	5	5	3	4	4	5	4	4	5	3	2
10	4	4	5	2	4	4	4	5	4	4	5	4
11	4	4	4	4	4	3	4	4	4	4	4	4
12	4	4	5	5	4	4	5	4	5	4	4	4
13	4	4	5	4	4	4	4	4	4	4	4	4
14	3-6	3-6	<3	<3	3-6	<3	<3	3-6	<3	3-6	<3	<3
15	-	-	-	-	-	-	-	-	-	-	-	-
16	-	-	-	-	-	-	-	-	-	-	-	-
17	-	-	-	-	-	-	-	-	-	-	-	-
18	4	4	4	4	4	4	4	4	5	5	4	4
19	4	3	4	4	4	4	4	1	5	5	4	4
20	5	5	4	4	4	3	4	1	5	5	3	4
20.1	5	5	5	4	4	4	4	4	5	5	3	4
21	5	5	5	5	4	4	4	2	5	5	4	5
22	5	5	5	5	4	4	4	2	5	5	4	5
22.1	5	5	5	5	4	4	4	2	5	5	4	5
23	3	5	5	4	4	4	4	2	5	5	4	5
24	5	5	5	4	4	4	4	2	5	5	4	5

Mts indicates months and N/A is not applicable.

Likert Scoring: Strongly Agree=5
Agree=4
Neutral=3
Disagree=2
Strongly Disagree=1

Except for items 9 & 10 when reversed scoring applies (eg: Strongly Agree=1,Agree=2,Neutral=3,Disagree=4,Strongly Disagree=5).

Please refer to separate sheets for qualitative data analysis of items 15,16 and 17.

QUALITATIVE DATA ANALYSIS OF DVT SCALE QUESTIONNAIRE.
(Trauma Directorate)

SECTION ONE: BIOGRAPHICAL DATA

1. How long have you been qualified?

One registered nurse was qualified for less than 6 months. The remaining eleven trained staff who participated in the data collection had varying levels of clinical experience, ranging from 2 to 33 years.

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2. What are your professional qualifications.

Staff in the study represented a good skill mix. One of the trained staff was a second level nurse with 21 years of experience and another was a first level nurse with 4 years experience in trauma / orthopaedics. The remaining 10 qualified staff were Nursing Diplomates with post-registration experience and qualifications awarded by the English National Board (ENB).

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3. What is your job title / clinical grade?

Six staff were employed in the clinical capacity of D grade, three as E grade and the other three staff held the F grade.
Within the clinical grading structure, post-holders in D grade are responsible for assessing care needs and the development of programme of care and or the implementation and evaluation of their programmes.
E grade applies to those who are responsible for the assessment of care and the development, implementation and the evaluation of programme of care and is designated to take regular charge of a ward. They are expected to supervise junior staff.
Additional to undertaking the role of D and E grade, F grade post-holders are required to lead a team of staff at grade E and below.

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4. How long have you been working in this area of practice?

Staff had worked on the trauma / orthopaedics directorate for varying period, ranging from less than 6 months for newly qualified staff to very experienced nurses with 18 years in this area of practice.

---

5. Have you worked in other clinical area(s) where the Autar DVT scale is being used?

None of the respondents had prior experience of using the DVT scale as none of them had worked in other clinical areas where this risk assessment tool was being used.

SECTION TWO

6. How many patients have you assessed with the DVT risk assessment scale?

During the data collection duration process of two months, staff had limited and varying experience of assessing patients with the DVT risk calculator. Six staff assessed less than five patients and the remaining staff recorded between five and fifteen patients.

7. The DVT scale enables me to:

7.1. make a thorough DVT risk assessment based on the patient clinical profile.

The DVT scale was positively evaluated by the majority of staff, enabling them to make a through DVT risk assessment. One respondent was neutral and another noted that the scale should be used in conjunction with patient assessment.

7.2. individualise the nursing assessment of patients.

There is consensus that the DVT scale individualised nursing assessment, identified patients into a particular risk category and allows for prophylactic anticoagulant and mechanical interventions to be initiated.

7.3. translate the risk factors as applicable to patients into DVT risk categories.

The respondents unanimously agreed that the risk factors could be construed to identify patients into DVT risk categories.

7.4. state why patients are at risk.

Nine respondents concurred that the DVT scale enabled them to understand why patients were at risk. One of the three neutral responses noted that the reason for patients at risk should be apparent to nurses

8. The DVT scale can be applied to facilitate care planning.

All respondents acknowledged that the DVT scale could be applied to facilitate care planning.

9. The DVT scale does not enable me to take preventative nursing action to minimise risk.

There were some mixed responses to this statement. The DVT scale highlighted the risk factors to consider and while anticoagulation prescription was the province of physicians, nurses were well placed to identify those are risk and make appropriate referral.

10. The DVT scale has no value as a teaching tool.

There was broad consensus that the scale was a valuable teaching tool. It enabled the staff to teach patients and their relatives of the risk factors of DVT and how to minimise risk.

11. The DVT scale has potential application as an audit tool for quality assurance.

The potential application of the DVT scale as an audit tool for quality assurance was fully endorsed by all the respondents. It could be used to monitor venous thromboprophylaxis protocol.

12. The DVT scale has practical application to my area of practice.

The incidence of DVT is notoriously high among trauma / orthopaedic patients and the respondents concurred that the DVT risk calculator had practical application to their speciality.

13. The layout of the DVT scale facilitates the data collection process for risk assessment.

It was commonly accepted that the easy to follow layout facilitated a quick DVT risk assessment.

14. The DVT scale enables me to complete a risk assessment in within < than 3 mins to > than 10 mins.

Its user-friendliness enabled staff to complete risk assessment within less than 3 minutes to 6 minutes. It appeared that the time taken by the staff was experienced related. Those who took less than 3 minutes to complete the risk assessment were senior clinical nurses and team leaders who occupied high position in the clinical grading structure.

15 Please comment on the strengths of the DVT scale.

The following qualitative comments about the DVT scale were reported:

- Sets out well, easy to read and score.
- Very good and reminds you of patients who are vulnerable.
- Well laid out.
- Simple to understand and complete.
- Virtually self-explanatory.
- Covers all categories.
- Identifies patients at risk.
- A good assessment tool.
- Easy to apply in any area of practice.
- Helps to take preventative measures to minimise risk.

16. Please comment on the weaknesses / limitations of the DVT scale.

The following qualitative records were noted in relation to the weaknesses and limitations of the DVT scale:

- Does not differentiate between male and female.
- Does not recommend what prophylaxis could be used in moderate and high risk patients.
- Needs a space to record daily changes in progress.
- Time consuming on an admission ward.
- Can't think of any.
- Needs a bit of background information with the scale and its application.

17 Any comments on how the practical application of the DVT scale can be improved.

The following suggestions were put forward to improve the practical applications of the scale:

- Can be integrated in a careplan already in use on the unit.
- Merged into Waterlow scale so that it can also be used to assess patients at risk of pressure sore.
- It could be laid out in a more linear fashion as the Waterlow scale.
- Not really, all aspects of risks have been covered.
- Included into an admission package or folder.

18. Age Specific Group:
This category is sufficiently clear to score.

The respondents unanimously agreed that the age specific group category was clear to score.

19. Mobility:

Scoring item from this category is reasonably clear.

Majority of the respondents found this category reasonably clear. One respondents was uncertain as to whether mobility related to pre-admission mobility or mobility status on admission.

20. Build:

I am able to differentiate between underweight and average build.

Most respondents were able to differentiate between underweight and average build. BMI ready reckoner facilitates the process when in doubt. One respondent strongly disagreed with this statement but provided no explanation.

20.1. I am able to differentiate the overweight patients from the obese and very obese.

Eleven participants were positively able to differentiate the overweight from the obese and very obese patients. One respondent was neutral but offered no reasons for the cause of the uncertainty.

21. Special risk category:

Although this category was not applicable to the area of practice, there was general agreement that it was clear to score.

22. Trauma risk category:

Please answer this question if this category is applicable to your area of practice.

I am reasonably clear as to what constitutes scoring for lower limb injuries.

This category was very relevant to the area of practice. Eleven respondents were clear as to what constituted scoring for the lower limbs. One respondent disagreed with the statement but offered no reason to justify disagreement.

23. Surgical intervention category:
Eleven respondents could differentiate between minor and major surgical intervention. The very same respondent to item 21 disagreed and offered no explanation

23.1. I am able to differentiate between major surgery and the other types of surgery.
The same response as item 22.

24. High risk diseases:

I have no difficulty scoring any item from this category:

The same response as items 22 and 22.1.

Appendix 15

Data Analysis: DVT scale questionnaires (Medical Directorate).
Total No of respondents: 8

RESPONDENTS

Items no	1	2	3	4	5	6	7	8
1	5Yrs	12yrs	1yr	17.5yrs	3yrs	3mts	4.5yrs	6yrs
2	RN	RN	DipN	RN FETC	DipN	DipN	DipN	DipN
3	E	E	D	G	F	D	F	E
4	3yrs	10mts	11mts	13yrs	3yrs	3mts	4.5yrs	3.5yrs
5	No	No	No	No	No	No	No	No
6	5-10	5-10	<5	5-10	>15	<5	<5	<5
7.1	5	4	5	5	5	4	5	5
7.2	4	4	4	5	4	4	5	4
7.3	5	5	4	5	4	4	5	4
7.4	4	5	4	5	5	4	4	4
8	4	4	4	5	4	4	4	4
9	4	5	4	5	5	4	4	4
10	2	3	4	5	4	4	4	3
11	4	4	3	5	5	4	5	3
12	4	4	4	5	5	-	5	4
13	5	4	4	5	5	3	5	4
14	<3mins	3- 6 mins	<3mins	<3mins	<3mins	<3mins	<3mins	<3mins
15	-	-	-	-	-	-	-	-
16	-	-	-	-	-	-	-	
17	-	-	-	-	-	-	-	-
18	5	4	4	5	5	4	5	4
19	5	4	5	5	5	4	5	4
20	4	4	4	5	5	4	5	4
20.1	4	4	4	5	4	4	5	4
21	5	4	3	5	N\A	N\A	5	4
22	5	4	4	5	N\A	N\A	5	N\A
23	4	4	4	5	N\A	N\A	5	N\A
23.1	4	4	4	5	N\A	N\A	5	N\A
24	5	4	4	5	4	4	5	4

Likert Scoring: Strongly Agree= 5
Agree=4
Neutral=3
Disagree=2
Strongly Disagree=1

Except for items 9 & 10 when reversed recording applies(eg: Strongly Agree=1, Agree=2, Neutral=3, Disagree=4, Strongly Disagree=5).

Please refer to freetexts comments for qualitative data analysis of items 15,16 and 17.

QUALITATIVE DATA ANALYSIS OF DVT SCALE QUESTIONNAIRE.
(Medical Directorate)

SECTION ONE: BIOGRAPHICAL DATA

1. How long have you been qualified?

Participants had a broad and varying range of experiences little as three months for a newly registered nurse to 17.5 years for the very experienced team leader.

---

2. What are your professional qualifications?

All participants were professionally registered nurses. Three of the participants were registered through the traditional 3-year nurse training at certificate level. The other five nurses had
undertaken the 3-year Diploma HE Nursing programme. Although the diploma nurses had less experience in terms of longevity, they were competent and " knowledgeable doer". Experience does not necessarily relate to the mere passage of time or longevity, but" rather the refinement of preconceived notions and theory through encounter with many actual practical situations that add nuances or shades to theory"(Benner 1984).

---

3. What is your job title / clinical grade?

Respondents belonged to different grades in the light of their clinical accountability and responsibility as defined by the national clinical grading structure. Two recently qualified staff were employed as D grade staff nurse. Other appointees included three E grade, two F Grade and the Ward manager / Team Leader at G grade level.

---

4. How long have you been working in this area of practice?

Newly qualified staff worked on the medical directorate for as little as 3 months and the most experienced staff for 13 years.

---

5. Have you worked in other clinical area(s) where the Autar DVT scale is being used?

None of the participants had prior experience of the practical application of the Autar DVT scale.

SECTION TWO

6. How many patients have you assessed with the DVT risk assessment scale?

Participants had varying amount of practical experience in terms of the application of the DVT scale to risk assessment.Five participants recorded less than five patients each and the remaining three staff risk assessed between five to ten patients.

7. The DVT scale enables me to:

7.1. make a thorough DVT risk assessment based on the patient clinical profile.

Responses were held from strongly agree to agree, unanimously concurring that the DVT scale configured a patient profile, which facilitated a through and comprehensive DVT risk assessment.

7.2. Individualise the nursing assessment of patients.

There was a strong consensus among the respondents that the DVT scale individualised risk assessment. It identified the nature of risk, derived from multifactoral aetiology and placed the patient into one of the risk categories. Individualisation of risk subsequently facilitated the application of the most appropriate intervention (Caprini et al,1991; Clagett et al,1992).

7.3. translate the risk factors as applicable to patients into DVT risk categories.

There was general agreement that the risk factors were readily construed into risk categories: the risk factors are additive and the aggregate score of the risk factors places patient into one of the risk categories within the assessment protocol.

7.4. state why patients are at risk.

It was generally accepted that the reasons for the patients being at risk of DVT were seamlessly made transparent by the DVT scale.

8. The DVT scale can be applied to facilitate care planning.

The respondents unanimously acknowledged the positive association between the DVT scale and care planning. It helped to" highlight certain issues and provides better clinical judgement to care planning".

9. The DVT scale does not enable me to take preventative nursing action to minimise risk.

All respondents disagreed with this statement and positively identified the value of the scale in terms of enabling them to take appropriate actions. It "serves as a reminder as to whether prophylaxis is in use or required".

10. The DVT scale has no value as a teaching tool.

Mixed responses were evident to this item. One respondent agreed with the statement but offered no supportive explanation. Five respondents valued the risk calculator as a teaching tool. Two staff gave neutral responses but no comments were offered to support their responses.

11. The DVT scale has potential application as an audit tool for quality assurance.

Six respondents concurred on the potential application of the DVT scale as an audit tool for quality assurance. Two neutral responses were also received.

12. The DVT scale has practical application to my area of practice.

There was consensus on the relevancy and practical application of the DVT scale to their area of practice.

13. The layout of the DVT scale facilitates the data collection process for risk assessment.

All respondents but one strongly agreed that the layout of the calculator eased the data collection process. It was described as " clear, precise, easy to understand and complete". The neutral participant offered no explanation as to her uncertainty.

14. The DVT scale enables me to complete a risk assessment in within < than 3 mins to > than 10 mins.

The " quick and easy to use features of the DVT scale enabled the participants to complete DVT risk assessment in less than 3 minutes.

15 Please comment on the strengths of the DVT scale.

The following freetext comments were noted:

- It enhances clinical assessment, highlighting areas that may otherwise be missed.
- Good.
- It identifies patients at risk, which may not have been realised until doing the risk assessment.
- Useful for those patients who don't appear to be at risk at first glance".
- Easy to use, not time consuming and steers towards the need for prophylaxis.
- Quick and self-explanatory.

16. Please comment on the weaknesses / limitations of the DVT scale.

The following freetext comments were recorded:

- It must not be used on its own but applied to support clinical knowledge and judgement.
- Need to get the doctor on board to ensure that high score patients have prescribed prophylaxis.
- It does not state previous MI in the high risk category.
- Very generalised.

17 Any comments on how the practical application of the DVT scale can be improved.

- Needs to be an integral part of patient assessment and not a stand-alone document.
- It is unclear that the risk factors in the high risk category imply previous medical history or current health status.
- "Hope this useful tool will become part of risk assessment in the same way the Waterlow has and that we will quickly see an impact on patient care and standards".

18. Age Specific Group:
This category is sufficiently clear to score.
All respondents affirmed that this subscale was clear to score.

19. Mobility:

Scoring item from this category is reasonably clear.
It was unanimously agreed that this category was clear to record.

20. Build:
I am able to differentiate between the underweight and average build.

Patients were not generally weighed or measured on the unit, although most respondents agreed that there was no difficulty in differentiating between the underweight and average build. One respondent was neutral.

20.1. I am able to differentiate the overweight patients from the obese and very obese.

All respondents were able to differentiate the overweight from the obese and very obese.

21: Special Risk Category.

Please answer this question if it is applicable to your patients.

This category is reasonably clear for me to score.
Two staff claimed that this category was not applicable, One was neutral and the other five agreed that this category was clear to score and should be included, as this risk factor was occasionally applicable to their patients.

22. Trauma risk category:

Please answer this question if this category is applicable to your area of practice.

I am reasonably clear as to what constitutes scoring for lower limb injuries.

Three respondents noted that this was not applicable to their area of practice although one staff observed that this category was equally appropriate as patients were often admitted from the Trauma Unit.

23. Surgical intervention category:
I am able to differentiate between minor and major surgery.

Responses were relatively similar to item 22.

23.1. I am able to differentiate between major surgery and the other types of surgery.

As item 23.

24. High risk diseases:

I have no difficulty scoring any item from this category:

There was broad consensus that there was no difficulty in scoring item(s) from this category.

Appendix 16

Data Analysis: surgical directorate
Practical application of the DVT scale

Questionnaire Item No	Respondent 1	Respondent 2
1	15 Years	15 Years
2	EN/ RGN/ENB qualifications	EN/RGN/ENB qualifications
3	F grade/ Team Leader	E Grade/ Senior Clinical Nurse
4	6 Years	13 Years
5	No	No
6	50 patients	50 patients
7.1	4	4
7.2	4	4
7.3	4	4
7.4	4	4
8	4	3
9	2	2
10	3	3
11	4	4
12	4	4
13	3	4
14	< 3 minutes	3-6 minutes
15	See separate sheet	See separate sheet
16	See separate sheet	See separate sheet
17	See separate sheet	See separate sheet
18	4	4
19	2	4
20	4	4
20.1	4	4
21	4	4
22	4	0 (not applicable)
23	5	4
23.1	5	4
24	5	5

Likert scoring: Strongly Agree: 5
Agree: 4
Neutral: 3
Disagree: 2
Strongly Disagree: 1

Reversed scoring order for items 9 & 10 (e.g.: Disagree: 4 and Strongly Disagree: 5.

QUALITATIVE DATA ANALYSIS OF DVT SCALE QUESTIONNAIRE. Surgical Directorate)

In the current cost cutting climate, staff shortage and plea for minimal imposition on nursing time, only the support of two registered nurses could be negotiated. They voluntarily and willingly offered to undertake the data generating activity on the targeted fifty patients on the surgical directorate. Two postal questionnaires mailed to them were duly received, evaluated and the following responses summarised:

SECTION ONE: BIOGRAPHICAL DATA.

1. How long have you been qualified?

The two nurses were senior clinical staff with fifteen years of post registration experience.

2. What are your professional qualifications?

Initially, both nurses commenced their professional career as level two Enrolled Nurses (EN) and successfully completed a conversion programme to register as level one nurse (RN). As part of their professional portfolio development, both registered nurses undertook validated English National Board (ENB) courses and gained appropriate post registration qualification.

3. What is your job title/clinical grade?

One nurse was grade F Team Leader Ward Sister) and her paired colleague, an E grade Senior Clinical Nurse (SCN).

4. How long have you been working in this area of practice?

Both nurses were experienced and competent clinical practitioners: the Team Leader worked in the surgical directorate for six years and the Senior Clinical Nurse had worked in the same area of practice for thirteen years.

5. Have you worked in other clinical area(s) where the Autar DVT scale is being used?

None of the two practitioners had any working experience of using the DVT scale.

SECTION TWO: THE AUTAR DVT RISK ASSESSMENT SCALE.

6. How many patients have you assessed with the DVT scale?

Both paired nurses independently assessed all fifty patients who were recruited to the study for DVT risk. As a result, both respondents developed good working experience of the scale and were in a position to make constructive feedback on the clinimetry of the DVT calculator as well as its practical application.

7: (7.1, 7.2, 7.3 & 7.4)

Evidently, there was good consensus that the clinical profile of the patient enabled the assessors to make a thorough and comprehensive assessment for risk of DVT as well as individualising the assessment process.

Additionally, there was strong agreement that assessment protocol of the DVT scale allowed the risk factors to be translated into main risk categories. Identification of patients in the main risk categories facilitates the choice of the most appropriate venous thromboprophylaxis (NIH, 1986, International Consensus Statement, 1997, THRiFT,1998). It was also acknowledged that reflecting on the risk factors for DVT enabled them to be watchful and "take a second look at patients for risk".

8.The DVT scale can be applied to facilitate care planning.

Mixed responses were received. One respondent acknowledged the potential of the DVT scale to facilitate care planning, while the other response was neutral. No comments were made on the choice of the neutrality of response.

9. The DVT scale does not enable me to take preventative nursing action to minimise risk.

Both respondents disagreed with this statement. In fact, this statement was deliberately structured in a negative overtone, to ensure that respondents to the Likert type scale questions do not respond passively in an automatic and central tendency mode (Oppenheim 1992). They actively affirmed that an awareness of the risk of DVT to a particular patient prompted them to initiate measures to minimise the risk.

10. The DVT scale has no value as a teaching tool.

Responses to the value of the DVT scale as a teaching tool were neutral. No comments were made as to the choice of the neutrality.

11. The DVT scale has potential application as an audit tool for quality assurance.

Respondents concurred that the DVT scale had potential for auditing performance. A formal assessment that helps to complete an audit cycle

improves the uptake of venous thromboprophylaxis (Bahal & Silverman,1993; Byrne et al,1996).

12. The DVT scale has practical application in my area of practice.

It was commonly acknowledged that the DVT scale has practical application to the surgical directorate. Surgical patients are a high risk group and with the exception of one, all forty-nine patients were recipients of pharmacological prophylaxis against DVT.

13. The layout of the DVT scale facilitates the data collection process for risk assessment.

Responses varied from being uncertain to complete endorsement of the layout of the DVT scale in relation to facilitating the data collection process and "is good as it is".

14. The DVT scale enables me to complete a risk assessment within less than 3 minutes to more than ten minutes.

Duration of DVT risk assessment with the risk calculator varied from less than 3 minutes to 3-6 minutes.

15. Please comment on the strengths of the DVT scale.

The following freetext comments were made:

" It gives clear indication of who may be at risk".

" It is a good way to double check if a patient is at risk".

" It makes you aware of who may be at risk".

16. Please comment on the weaknesses / limitations of the DVT scale.

It was viewed by one respondent as "yet another form to fill", noting that this was essentially a data generating study.

Another respondent suggested that the mobility subscale of the chart be more explicit. Complete bedrest as a risk factor weighted at a score of 4 could be further reviewed to indicate its duration in terms of days and weeks as some surgical patients were unavoidably confined to bed for long period following some major surgical intervention

18. This category is sufficiently clear to score.

The two respondents concurred that this risk subscale was easy to score.

19. Scoring item from this category is reasonably clear.

There was some disagreement between the two respondents on ringing out this risk category, albeit scoring and a calculated kappa statistics of 0.97 clearly indicated excellent consistency. While one respondent agreed that this category was transparent, this view was not shared by her colleague, suggesting that mobility as a category was not explicit enough, in terms of its temporality. This issue recurred in item 16 of the questionnaire.

20.Build. I am able to differentiate between underweight and average build.

Both nurses agreed that they were able to differentiate between the underweight and average build patient as confirmed in the recording of this subscale.

20.1. I am able to differentiate the overweight patients from the obese and very obese.

Both respondents ably differentiated between the overweight, obese and very obese as demonstrated in the recording of this category. Additionally, if in doubt as to what is the most appropriate type of build the BMI chart on the reverse of the DVT scale offered practical guide to recording this risk category.

21. Special risk category. This category is reasonably clear for me to score.

Both respondents agreed that this risk category is clear, although this item was not applicable to their area of practice.

22. Please answer this question if this category is applicable to your area of practice.

One nurse who previously worked on the orthopaedic unit confidently commented that this was clear, although no such patient was recruited to the study. Her paired colleague had no such previous background and thus was unable to comment on this item.

23. I am able to differentiate between minor and major surgery.

Responses varied from strongly agree to agree. Major surgery is defined as a procedure on a patient requiring more than thirty minutes of general anaesthesia (Goucke, 1989; Moser, 1989).

23.1. I am able to differentiate between major surgery and the other types of surgery.

Responses varied from strongly agree to agree. Inter-observer consistency was evident.

24. High risk diseases.

I have no difficulty scoring any item from this category.

No inter-observer variation was demonstrated and extent of agreement was expressed in perfect qualitative term in the risk assessment process.